PAGE
189
191
193
201
212
234
235
237
239
241
245
260
276
281
281
283
284
289
353
291
305
307
315
328
336
340
344
355
370
375
388
391

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HÆMANGIOMATA ARISING IN THE BONES OF THE SKULL

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HÆMANGIOMATA arising primarily in bone are extremely rare; in the bones of the skull, not more than 31 have hitherto been described.

The 5 cases about to be recorded are of interest, not only because they represent the largest single series which has so far been reported, but because it has been possible, by personal observation, to compare their clinical and pathological features with those of osteomata, cholesteatomata, and meningiomatous hyperostoses.

The first accurate account of a hæmangioma arising primarily in the skull was given by Toynbee (1847) in a paper read before the Royal Medical and Chirurgical Society in London, in which he described two tumours found in the parietal bones of a young man who had died of consumption. These tumours consisted of a network of blood-vessels similar to that seen in a hæmangioma of the soft tissues. Further instances were recorded by Cruveilhier (1849), von Rokitanski (1856), Billroth (1856), Sangalli (1860), and Virchow (1867).

At the beginning of the present century, Schöne (1905) reviewed 7 cases which he had found in the literature and described a cavernoma growing in the occipital bone. In 1901, Zajaczkowski removed a hæmangioma arising in the frontal bone, including the underlying portion of dura mater to which the tumour had become adherent.

The first X-ray photographs of a hæmangioma arising in bone appeared in an article by Hitzrot (1917), who described the radiographic appearances of a hæmangioma growing in the upper end of the humerus of a young physician. In his comments, Hitzrot expressed the opinion that similar tumours in the skull probably arise either in the centres of ossification or near the suture lines.

Six years later, Cushing (1923) described the successful removal of a cavernous hæmangioma by block resection.

In a comprehensive paper, Bucy and Capp (1930) discussed the radiological and pathological features of 1 capillary and 7 cavernous hæmangiomata occurring in various bones of the body; of these, one only was recorded in the skull, and this had already been described by Cushing.

Anspach (1937) attempted to remove a large parietal-bone hæmangioma but had to abandon the attempt in the early stages, owing to severe hæmorrhage. This proved to be an important case, since it clearly illustrated the non-malignancy of calvarial hæmangiomata; fifteen years after the attempted removal, the tumour showed no evidence of intracranial extension or metastatic deposits.

Guided by the experience of others, successful resections have been accomplished by Dikansky (1932), Overend (1933), Mazzini and Brachetto-Brian (1936), Abbott (1937), Pich (1938), Porta and Clivio (1938), Sommer (1938), Kaplan and Kanzer (1939), and Jefferson (1941).

CASE REPORTS

Case 1.—**Pains in the head. A swelling in the right frontal bone.**

HISTORY.—A young woman, aged 32, was sent to the Christie Hospital by Dr. Hamilton, of Golborne, on Nov. 18, 1938. For eleven months she had experienced a dull ache over the right side of the forehead, associated with stabs of sharp pain. A small swelling on the forehead, noticed seven months previously, had slowly increased in size up to the time of the examination.

ON EXAMINATION.—On the right side of the forehead, just outside the hair line, was a small bony hard swelling attached to the frontal bone. It was roughly hemispherical in shape and projected half an inch, its base measuring 1½ in. in diameter. The skin moved freely over it; the cutaneous vessels were not enlarged and there was no pulsation or bruit. No other swellings were found elsewhere in the body. General examination was negative and there were no abnormal neurological signs; the special tests were negative. Radiography showed a circular defect, measuring

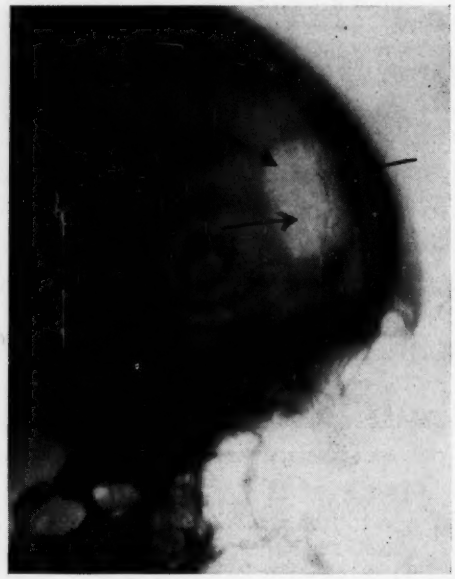
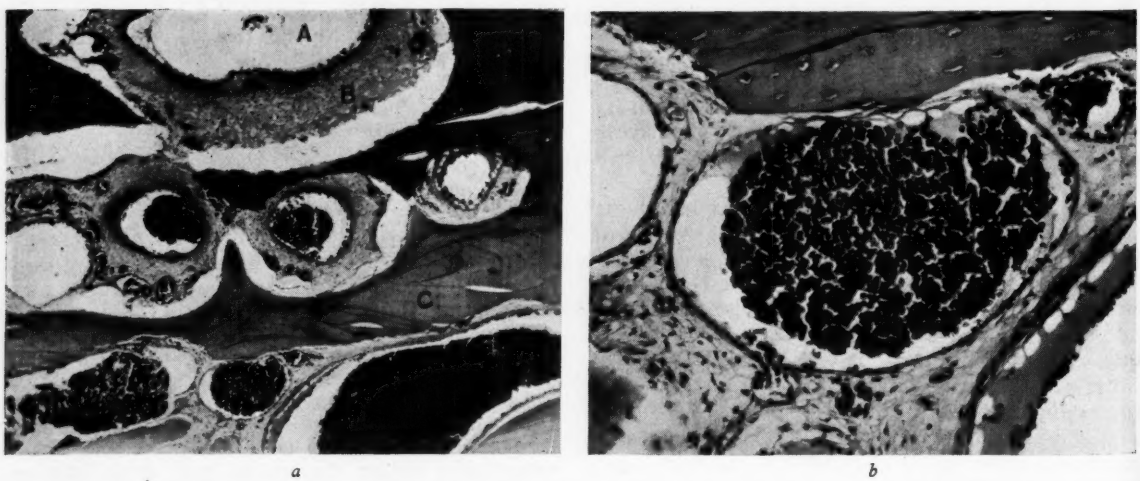


FIG. 1.—*Case* 1. Radiograph showing trabeculated erosion in the frontal bone. Lateral view.

FIG. 2.—*Case* 1. An X-ray view made from a cross-section of the removed hæmangioma. It shows a mound of newly-formed cancellous bone beneath the pericranium and enlarged vascular channels within the diploe. The inner table has not been depressed to any appreciable extent.

FIG. 3.—*Case* 1. A cross-section of the freshly removed tumour. (*Drawn by Miss Davison.*)



a *b*

FIG. 4.—*Case* 1. Microphotographs of the tumour, showing the typical appearance of a calvarial hæmangioma. A, Blood-spaces; B, Matrix; C, Bone. (*a*, × 20; *b*, × 75.)

1¼ in. in diameter, in the right frontal bone; the margins were clean cut and enclosed a network of fine bony trabeculæ (*Fig.* 1).

A pre-operative diagnosis was made of a hæmangioma of the frontal bone.

OPERATION, Dec. 29, 1938 (G. F. R.).—After a suitably curved skin flap had been turned, a small bluish swelling was seen beneath the pericranium. A large branch of the supra-orbital nerve was attached to the summit of the swelling, on a filament of which a small thickening was observed. This thickening was later shown by histology to be a neuroma.

The swelling was not pulsatile, but a swirl of blood could be plainly seen in the region of its summit. By means of a large trephine, the swelling and its covering membrane were removed in the form of a disc. The underlying dura mater was normal in every way and there was no evidence of an intradural tumour. The defect in the skull was repaired by filling in the hole with a trephine disc taken from the tibia.

Post-operative convalescence was uneventful. Within two months the girl was back at work and no deformity of the skull was evident.

PATHOLOGY OF THE SPECIMEN.—

Macroscopical.—The specimen consisted of a trephine disc. A cross-section showed that the inner table was not depressed and that the diploe consisted of a mass of large spaces containing bright red blood. There was a mound of cancellous bone under the pericranium. (*Figs.* 2, 3.)

Histological.—The trephine disc was sawn into thin slices, fixed in 10 per cent formol saline, decalcified in nitric acid, and embedded in paraffin. Sections were stained with hæmalum and eosin and by the Mallory and van Gieson techniques. Microscopically, the specimen consisted of numerous blood-spaces embedded in a matric of fibrous tissue, enclosed by a framework of bony trabeculæ. Flattened cells, presumably of an endothelial nature, lined the blood-spaces. No smooth muscle or elastic tissue was found in their walls. The supporting matrix, consisting of connective tissue of variable cellularity, contained small islands of myelogenous tissue, no doubt the remnants of the original diploe. (*Fig.* 4).

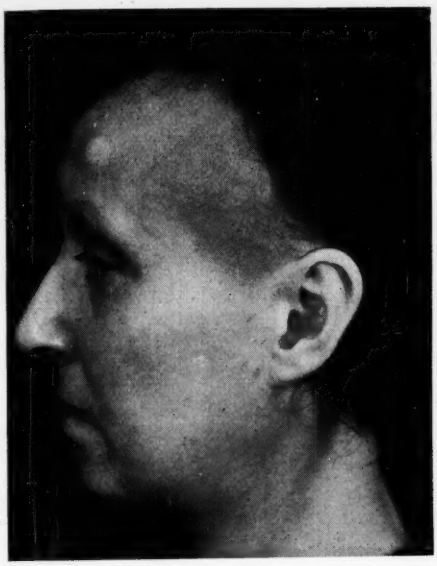


FIG. 5.—*Case* 2. Photograph showing the small swelling on the left side of the forehead.

Case 2.—**A swelling on the left side of the forehead, associated with neuralgic pains and headaches.**

HISTORY.—A woman, aged 49, was admitted to the Neurosurgical Service of the Manchester Royal Infirmary, under the care of Mr. G. Jefferson, in February, 1934. Eighteen months previously she had knocked her head rather badly; a small lump appeared at the site of the bruise and this had steadily increased in size (*Fig.* 5). She complained of sharp pains over the swelling and persistent headaches of a generalized kind.

FIG. 6.—*Case* 2. Radiograph showing a benign erosion of the left frontal bone traversed by bony trabeculæ.

ON EXAMINATION.—On the left side of the forehead, projecting half an inch from the frontal bone was a bony-hard hemispherical swelling about 1 in. in width at its base. The skin moved freely over it, the overlying cutaneous vessels were not dilated, and there was no pulsation or bruit. No other swellings were found. General examination was negative and there were no abnormal neurological signs. The special tests were negative. Dr. Twining's report on the radiographs was: "There is a localized area of bone erosion with sharply defined margins. Within this area the bone is not completely destroyed, but has a finely trabeculated structure (*Fig.* 6). A diagnosis of a benign erosion is suggested by these

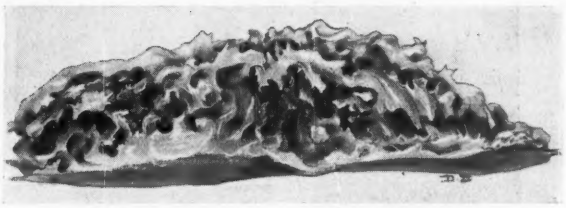


FIG. 7.—*Case* 2. Drawing of the removed tumour made under the dissecting microscope. It shows the honeycombed appearance of a diploic hæmangioma. (*Drawn by Miss Davison.*)

appearances, and the swelling possibly is a hæmangioma ".

OPERATION, March 2, 1934 (G. F. R.).—A transverse incision was made over the swelling. No abnormal vessels or adhesions were encountered and a blue-coloured non-pulsatile swelling was seen

FIG. 8.—*Case* 3. Photograph showing a swelling protruding from the upper margin of the right orbit.

beneath the pericranium. The swelling was removed quite easily by means of a large trephine and the underlying dura mater was seen to be perfectly healthy. The wound was closed in the usual way. Convalescence was uneventful. The neuralgic pain was relieved, but the headaches remained. When examined four years later, there were no signs of recurrence.

PATHOLOGY OF THE SPECIMEN.—

Macroscopical.—The specimen consisted of a trephine disc (*Fig.* 7). The inner table was thin and quite intact; the diploe was widened and the outer table was lifted into a dome-shaped swelling.

Microscopical.—The trephine disc was sawn into thin slices; these were fixed in 10 per cent formol

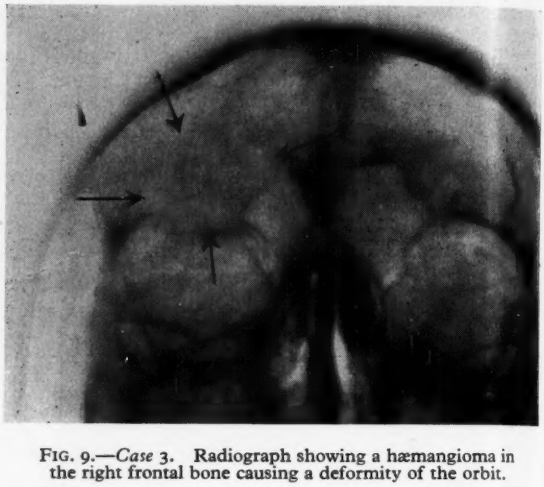
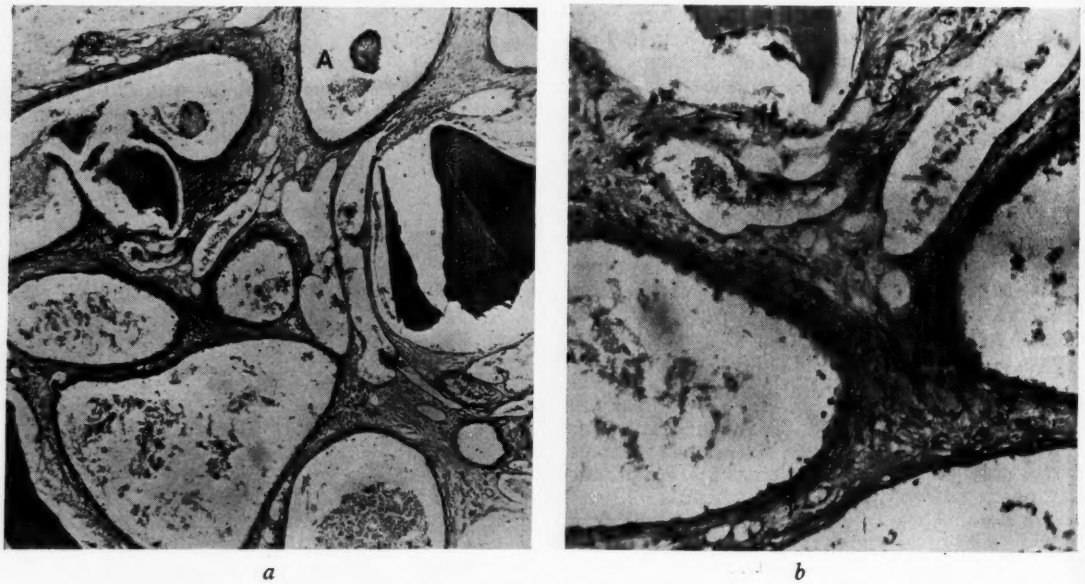


FIG. 9.—*Case* 3. Radiograph showing a hæmangioma in the right frontal bone causing a deformity of the orbit.

saline, decalcified in nitric acid, and embedded in paraffin. Sections were stained with hæmalum and eosin, and by the Mallory and van Gieson techniques. Microscopy showed large blood-spaces lined by flattened cells embedded in a matrix of connective tissue, the whole being enclosed in a framework of cancellous bone of adult structure. The tumour was obviously a hæmangioma of the cavernous type, but in this case was entirely diploic in position, and not, as in *Case* 1, partly subpericranial and partly diploic.



a *b*

FIG. 10.—*Case* 3. Microphotographs of the hæmangioma. A, Blood-spaces; B, Matrix; C, Bone. (*a*, × 16; *b*, × 60.)

Case 3.—**A swelling in the roof of the orbit, causing ptosis and displacement of the eyeball, associated with headaches.**

HISTORY.—A woman, aged 66, was admitted to the Neurosurgical Service of the Manchester Royal Infirmary, under the care of Mr. G. Jefferson, in January, 1934. Five years previously she had received a severe knock on the right side of her face; six months later she noticed that the eyelid was beginning to droop, and that her eye was being pushed downwards (*Fig.* 8). From this time onwards, she had suffered persistent pains about the orbit and had been troubled with frequent headaches.

ON EXAMINATION. — There was a bony-hard non-pulsatile swelling, projecting half an inch from the outer half of the upper margin of the right orbit, extending backwards beyond the limits of palpation. The right eyelid was drooped. There was no paralysis of its muscles. The eye was displaced downwards. There were no other abnormal findings. The special tests were negative. Dr. Twining's report on the X-ray films was: "Above the right orbit is an area of altered bone; the upper margin of the orbit is depressed and the frontal air sinus deformed. The eroded area shows a fine trabeculation, caused probably by a network of circular vascular channels". (*Fig.* 9.)

FIG. 11.—*Case* 4. Radiograph showing benign erosion of bone due to a hæmangioma.

OPERATION, Feb. 5, 1934 (G. F. R.).—A curved incision was made along the line of the eyebrow; no abnormal vessels or adhesions were encountered and a nodular swelling covered by periosteum was seen projecting downwards from the roof of the orbit. The cortex of the bone was broken into by a chisel, and a highly vascular tumour was immediately exposed. Brisk hæmorrhage was encountered, but this was readily controlled by plugging the cut surface with wax. As it was impossible to excise the growth by block resection, it had to be removed piecemeal by means of nibbling forceps. The post-operative recovery was uneventful; the axis of the eye returned to a nearly normal position and the pains about the orbit were considerably relieved. Four years later, the patient was very well, apart from occasional headaches, and there was no evidence of recurrence.

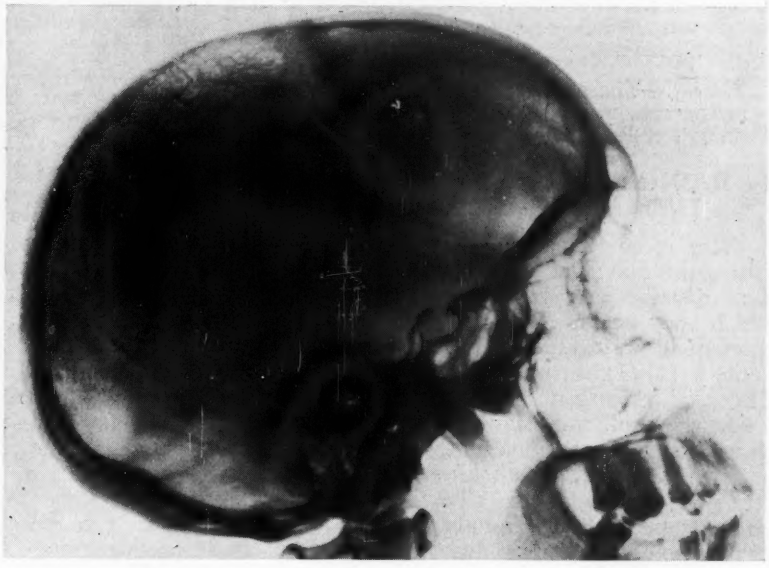


FIG. 12.—Typical 'sunburst' appearance of a hæmangioma of the calvarium.

PATHOLOGY OF THE SPECIMEN.—The tumour was a cavernous hæmangioma and was similar in histological details to those described in *Cases* 1 and 2 (*Fig.* 10).

Case 4.—**A swelling over the left frontal bone, associated with headaches.**

HISTORY.—A woman, aged 37, was admitted to the Neurosurgical Service of the Manchester Royal Infirmary, under the care of Mr. G. Jefferson, in June, 1941. Two months previously she had noticed a swelling on her head on the left side of the forehead. This had slowly increased in size up to the time of examination. She complained of occasional headaches.

ON EXAMINATION. — A hemispherical swelling about the size of a large button, obviously attached to the underlying bone, was found just above the left eyebrow. X rays showed a benign erosion of bone, enclosing fine bony trabeculæ (*Fig.* 11). A pre-operative diagnosis of hæmangioma was made.

OPERATION, June 14, 1941 (G. J.).—Local anæsthetic was injected along the left supra-orbital margin. The incision extended from one extremity of the supra-orbital margin along the eyebrow to the midline and then for 1 in. vertically. A flap of scalp was turned upwards to expose the tumour, which was found to be about 1 in. in diameter, well-defined and circular. It presented a bluish appearance when

intact. It was chiselled out with comparatively little bleeding and showed the typical honeycombed appearance of an angioma. The excision extended down to the dura, which was left intact and practically all the tumour was removed leaving a circular hole. A trephine disc was cut from the right ilium and hammered into the hole. It fitted perfectly. The wound was then closed, after dusting with powdered sulphanilamide.

Case 5.—The only records in this case consist of a radiograph, which was found in the late Dr. Twining's collection (*Fig.* 12).

RADIOLOGICAL FEATURES OF HÆMANGIOMATA IN BONE

The radiological features of hæmangiomata arising in bone vary according to the stage of their development, and to the type of bone in which they occur. They may be classified into three main groups: (1) Those of the skull or flat bones; (2) Those of the long bones; and (3) Those of the spinal column.

In the skull they show as local erosions with clearly defined margins enclosing a meshwork of fine trabeculæ, which often radiate from the centre of the tumour to give the classical 'sunburst' appearance commonly associated with the hæmangiomata. This radial arrangement of the trabeculæ, however, does not occur in all cases, as illustrated by *Figs.* 1, 6, 9, 11, in which the trabeculæ are irregularly disposed.

In the long bones, hæmangiomata usually show as cystic swellings enclosing stout trabeculæ, and occur especially in the diaphysis near to the epiphysial line. In the vertebræ, the bone loses its homogeneous structure, and becomes striated in a vertical direction, with alternating bands of rarefaction and increased density. These changes are best seen in the bodies, but the pedicles, laminæ, and spines may also be affected.

PATHOLOGY

Although four of the tumours on which this paper was based were solitary—as far as could be judged without a full post-mortem examination—hæmangiomata are commonly multiple. In the fifth case, radiological details only were known.

From the following table, it will be seen that, of the 31 cases of hæmangiomata found in the skull, 6 were in the frontal bone, 12 in the parietal bone, 4 in the occipital bone, and 2 in the petrous bone. In 7 cases the position of the tumour was not stated. The ages of the patients ranged from 3 to 76 years, and in 26 cases where the sex was stated, 9 occurred in men and 17 in women.

DETAILS OF 31 RECORDED CASES OF HÆMANGIOMATA OF THE SKULL

NAME OF AUTHOR	YEAR	AGE	SEX	SITE OF TUMOUR	TREATMENT	RESULT	PATHOLOGY OF TUMOUR	REMARKS
1. Toynbee	1847	19	M.	Both parietal bones	Autopsy	—	Hæmangioma	Patient died of consumption
2. Cruveilhier	1849	—	F.	12 tumours in skull and 4 other tumours in the body	Autopsy	—	Cavernous tissue filled with liquid blood	—
3. v. Rokitansky	1856	—	—	Parietal bones	—	—	Cav. blood tumours	—
4. Billroth	1856	—	—	Cranial bones	Autopsy	—	Cav. blood tumour	Tumours in cheek, liver, spleen, and cranial bones
5. Sangalli	1860	—	M.	Occipital bone	Autopsy	—	Cav. hæmangioma	Death due to pulmonary disease
6. Virchow	1867	72	F.	R. parietal bone	Autopsy	—	Hæmangioma	An angioma was also present in the liver
7. Morris	1880	40	F.	R. parietal bone	Punctured	—	Tumour had begun in diploe and extended inwards and outwards	—
8. Pilcher	1894	15	F.	Frontal bone	Piecemeal excision	Recovery	Hæmangioma of diploe	History of trauma
9. Ziegler	1901	—	—	Vault of cranium	—	—	Probably a hæmangioma	A drawing only
10. Politzer	1901	12	F.	R. petrous bone	Autopsy	—	Cav. hæmangioma	—
11. Zajaczkowski	1901	38	F.	Frontal bone	Excision	Recovery	Cav. hæmangioma	Tumour was adherent to dura and a piece of this membrane had to be excised
12. Schöne	1905	39	M.	L. occipital bone	Excision	Recovery with relief of headache	Cav. hæmangioma	A good historical review is given of the early cases

DETAILS OF 31 RECORDED CASES OF HÆMANGIOMATA OF THE SKULL—*continued*

NAME OF AUTHOR	YEAR	AGE	SEX	SITE OF TUMOUR	TREATMENT	RESULT	PATHOLOGY OF TUMOUR	REMARKS
13. Major and Black	1918	34	M.	L. frontal and R. temporal bones	—	—	Cav. hæmangioma	Death due to angioma in liver. Both adrenals were cystic
14. Kaufmann	1922	76	M.	Skull	Autopsy	—	Cav. hæmangioma	Angiomas in liver and other organs
15. Cushing	1923	55	F.	L. parietal bone	Block resection	Cure	Cav. hæmangioma	This case was described later by Bucy and Capp
16. Eros	1928	70	F.	R. frontal bone extending to L. frontal bone	Autopsy	—	Simple cav. hæmangioma	Died from other causes
17. Eigler	1930	62	M.	Several angiomas in skull	Autopsy	—	Cav. hæmangioma	—
18. Dikansky	1932	20	M.	L. temporal and parietal region	Resection	Recovery	Cav. hæmangioma	History of trauma
19. Dikansky	1932	17	F.	R. parietal bone	Resection	Recovery	Cav. hæmangioma	Injury sixteen years before
20. Overend	1933	28	M.	Occipital bone	Resection	—	Cav. hæmangioma	Tumour grew inwards as well as outwards
21. Reischauer	1933	40	F.	Parietal bones	Specimen	—	Cav. hæmangioma	—
22. Mazzini and Brachetto-Brian	1936	39	F.	R. frontal bone	Resection	Recovery	Cav. hæmangioma	History of trauma
23. Geschickter	1936	—	—	Angiomas of skull	—	—	—	5 cases are mentioned but 3 at least are very doubtful
24. Geschickter	1936	—	—	Angiomas of skull	—	—	—	—
25. Abbott	1937	35	F.	R. frontal region	Resection	Recovery	Fibrous angioma filled with blood	—
26. Anspach	1937	11	F.	L. parietal bone	—	Patient well 15 years later	—	A series of excellent radiographs are shown
27. Porta and Clivio	1938	44	F.	R. parietal bone	Excision	Recovery	Hæmangioma	—
28. Pich	1938	44	M.	L. parietal bone	Excision	Recovery	Cav. hæmangioma	Author calls it an osteoangioma
29. Sommer	1938	16	F.	Occipital region	Excision	Recovery	Cav. hæmangioma	—
30. Kaplan and Kanzer	1939	47	F.	L. parietal bone	Block resection	Cure	Hæmangioma	The pathology of the tumour was diagnosed pre-operatively
31. Mangabeira-Albernaz	1939	21	F.	Middle ear and temporal and petrous bone	Autopsy	—	Cav. hæmangioma	—

Of the benignity of hæmangiomata arising primarily in the bones of the skull there is no doubt whatever, as shown by Anspach's case and by two of my own cases, in which there was no evidence of recurrence four years after the most conservative excisions. Also, the tumours show little tendency to grow inwards to compress the brain. Metastatic deposits in lymph-glands are unknown.

Cases have been described in which the dura, as well as the bone, has been involved in a hæmangiomatous mass, but such tumours are quite distinct from the purely diploic hæmangiomata. In these cases, the neoplasm probably arises in the dura, invasion of the bone being secondary.

Malignant tumours of the skull, such as sarcomata or angio-endotheliomata, may be extremely vascular and mimic the benign hæmangiomata. They can, however, be easily differentiated by their rate of growth and by histological examination.

Increased vascular marking of the skull, caused by angioblastic meningiomata, and cirsoid aneurysms of the scalp or brain, are diffuse and in no way resemble the localized erosions of diploic hæmangiomata.

Calvarial hæmangiomata are usually of the cavernous type. They consist of large dilated blood-spaces embedded in a connective tissue of variable cellularity, and they are lined by long flattened cells. Elastic or muscle tissue, such

as occurs in the walls of normal blood-vessels, is never seen in calvarial hæmangiomata. The bony trabeculæ in which the tumour is enclosed are not neoplastic in nature, as shown by their continuity with the surrounding skull and by the orderly arrangement of their lamellæ. The honeycombed structure of the bone is due to remodelling, and is brought about by the physiological process of bone formation and bone destruction.

DIFFERENTIAL DIAGNOSIS

Hyperostoses, resulting from invasion of the calvarium by meningioma cells, are invariably associated with intracranial extensions, giving rise either to local neurological signs or to raised intracranial pressure. Also, the protein content of the cerebrospinal fluid is often raised from the normal of 35 mg. to about 100 mg. per cent. Radiologically, the picture is usually that of a diffuse sclerosis, wide areas of the bone undergoing complete architectural remodelling (Rowbotham, 1939).

Occasionally a complete and local destruction of bone occurs—osteoclastic hyperostosis. In this case the picture is that of a thin dome-shaped shell of bone rising from the outer table, being distinguishable from a hæmangioma in that it does not enclose trabeculæ.

A *cholesteatoma* arising in the bones of the calvarium may or may not be associated with signs of intracranial extension ; usually it is not. Clinically, it appears as a hemispherical swelling attached to the skull of a bony-hard consistency. Radiologically, itshows as a benign erosion with densely sclerosed margins, trabeculation being absent.

An *ivory osteoma*, clinically, is indistinguishable from a calvarial hæmangioma. Radiologically, it shows as a dense homogeneous shadow projecting from the outer table of the skull. The tumour is often pedunculated or mushroom in shape.

A calvarial hæmangioma occurs as a bony-hard slowly-growing hemispherical swelling. It never gives rise to intracranial complications, but may be associated with headaches and pain. Its diagnosis depends purely on its radiological features. It shows as a benign erosion enclosing fine bony trabeculæ which may or may not be disposed radially in the typical ' sunburst ' fashion.

I should like to take this opportunity to thank Professor Jefferson for the generous use of his material which is incorporated in this article.

Also I should like to express my thanks to Mrs. Kenneth Watkins for the help she gave me in the review of the literature on this subject.

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FATIGUE FRACTURE OF THE TIBIA

By J. BLAIR HARTLEY, Manchester

I first noticed this rare and interesting condition in the left tibia of a boy aged 18 years sent complaining of pain and whose doctor suspected osteomyelitis.

The radiographic appearances were unusual and he was kept under observation for many months. I saw no similar cases, despite a careful watch through hundreds of casualty and orthopædic clinic cases until April, 1932, when the same appearances were discovered in the same situation in the left tibia of a girl of 16 years of age. Since then I have detected 12 more cases of this type.

The patients complain of pain in the calf, or near the upper end of one tibia, and generally to the inner side. There is little to see or feel, and X-ray investigations are invoked. These reveal appearances which, in an early case, suggest that there may have been an incomplete fracture about 2½ in. to 3 in. below the knee-joint, involving the tibia only. A small knuckle of callus will usually be visible at this level, either on the inner aspect or posteriorly.

Within a few days there will appear either ill-defined osteosclerosis, or, if the case is a more severe one, an area of osteoporosis with its base posteriorly. There will be no abnormality in the fibula.

Knowing that this site is a most unusual one in which to find true fracture of the tibia, the radiologist must always examine the opposite leg. In 3 out of 14 cases, the condition was found in both tibiæ. Bilateral findings can lead to one diagnosis only.

Should a true fracture be diagnosed in this area in a person between the age of 7 and 20 years, one should immediately re-examine the films to make sure that the fracture has not occurred through a pathological area. If callus is visible at the time at which the fracture is diagnosed, or within a few days, one may be quite certain that the fracture occurred through pathological bone, and the next move should be to try to establish the diagnosis of pre-existing 'pseudo-fracture'.

The reason for publishing this survey is that one of the most recent cases has demonstrated the need for widespread recognition of the existence of this condition during war, for in this instance the failure of diagnosis led to severe fracture of tibia and fibula, and consequently to prolonged and unnecessary immobilization of a recruit to a fighting regiment.

Now that younger age-groups are liable for war service, the incidence is likely to be greater, since fatigue-fracture is more common in adolescence.

I prefer the term 'fatigue fracture' to 'pseudo-fracture', for the latter suggests that the condition is not really a fracture, whereas we now know that it should in fact be regarded as a type of fracture which under certain circumstances may lead to infraction. The pseudo-fracture group may *include* fatigue fractures, but not all pseudo-fractures are fatigue fractures (e.g., Looser's zones in osteomalacia).

I have been unable to find any reference to this condition in the literature prior to November, 1939, when Roberts and Vogt[1] described 12 cases.

In their series there was one bilateral example; in my series there were three.

Tables I and II show the incidence rate, together with the sex and age incidences in the author's cases as compared with those recorded by Roberts and Vogt.

Table I.—Fatigue Fractures: Present Series of Patients

Case No.	Patient First Seen	Age	Sex	Leg Involved	Distance from Knee-joint To Centre of Lesion
		yr.			in.
1	Dec., 1931	18	M.	Left	3
2	April, 1932	16	F.	Left	2
3	Aug., 1932	13	F.	Left	3½
4	Feb., 1936	15	M.	Bilateral	3
5	Nov., 1936	8	M.	Left	2½
6	Oct., 1936	11	M.	Left	3
7	June, 1938	16	M.	Right	3¾
8	Jan., 1939	14	M.	Left	2½
9	May, 1939	15	M.	Bilateral	3
10	Sept., 1938	7	M.	Right	2¾
11	Sept., 1940	14x	M.	Right	3½
12	Nov., 1940	20	M.	Bilateral	3¾
13	Feb., 1941	17	M.	Right	3
14	May, 1941	8	F.	Left	2

Table II.—Fatigue Fractures: Roberts and Vogt's Series

Case No.	Patient First Seen	Age	Sex
		yr.	
1	July, 1931	12	M.
2	Dec., 1933	6½	F.
3	Dec., 1933	11 5/12	M.
4	Dec., 1934	8½	M.
5	Dec., 1935	15	M.
6	Dec., 1935	9½	M.
7	Nov., 1936	15	M.
8	May, 1937	13	M.
9	March, 1937	10	M.
10	Nov., 1937	4	F.
11	Oct., 1937	10½	M.
12	July, 1938	16	M.

Kohler[2] refers to "indirect fracture" at the junction of the upper and middle thirds of the fibula, and mentions 30 cases seen "in a few

years " by a Dr. Glogan. It is interesting to note that these fractures, occurring without history of violence, are there referred to as " recruit's disease ".

H. Jackson Burrows[3] recently described 2 apparently similar cases occurring at the junction of the middle and lower thirds of the fibula under the title of " spontaneous fracture of apparently normal fibula in its lower third ". In his discussion he said everything except actually to state that these were ' fatigue fractures ' ; for there is certainly great similarity between his 2 cases and the condition to be described here.

Weaver and Francisco[4] recently reported 2 cases of pseudo-fracture of the tibia (one bilateral) and 1 case of pseudo-fracture of the lower end of the fibula. These cases dovetail neatly with those simultaneously reported by Jackson Burrows.

McCullough and Macey[5] support my contention below that the condition here described as fatigue fracture is distinct from pseudo-fracture, which is a term which might well be kept to *include* the infractions and *Umbauzonen* seen in : (1) Paget's disease ; (2) Osteomalacia (Looser's zones) ; (3) Osteogenesis imperfecta tarda ; (4) Fatigue fracture ; etc. It is confusing the issue to have a name which means different things to different observers. Fatigue fracture, I maintain, is a distinct condition and must be kept separate at least whilst the literature on this clinical and radiological condition is becoming stabilized.

For example, Macey[5] reports a case of multiple " pseudo-fractures ", which is the exact antithesis of any case seen or described by myself, or described by Roberts and Vogt in the original paper. It differs also from Burrows' and from Weaver and Francisco's cases, precisely because : (*a*) there is multiplicity ; (*b*) the illustration shows a zone right across the femur. In none of our cases does the zone involve the whole width. In all our cases the site is at the point of maximum strain.

CLINICAL FEATURES

Generally there is no history of injury, certainly there is never a history of violence likely to cause true fracture at the affected site. In 14 cases incidence of reported injury was as follows : (*a*) 9 patients, *no* history of injury ; (*b*) 4 patients, a history of a fall or blow occurring within the previous week or two ; (*c*) 1 patient, no definite history of injury—recorded in clinical notes as " thought to have fallen ".

The only constant symptom is pain on standing, on walking, or after walking. This may be either behind the knee, in the calf, or over the inner aspect of the upper third of the tibia. There is no pain when the leg is at rest. So far no bilateral case has complained of pain in both legs simultaneously.

There has been no record of rise of temperature.

In 5 patients only there was slight swelling to be detected on the inner aspect of the upper end of the shaft of the tibia.

The case histories record no erythema, even in those patients in whom swelling was noticed.

Tenderness was recorded only in 3 instances.

The usual period, in my series, from the onset of symptoms to radiographic examination was from 10 to 21 days. In my opinion this condition in the tibia always occurs in children or in adolescents, the oldest case in my series being 20 years of age. The average age in the Roberts and Vogt series was 10·9 years ; the average age in my series was 13 years. The difference in the average age incidence is probably accounted for by the greater variety of nationalities met with in America, and by the fact that some of the races probably tend to mature at a slightly earlier age than is the case in this country. In every case there has been spontaneous cure, nothing more than rest from weight-bearing being necessary to produce this result.

RADIOLOGICAL FINDINGS

Radiologically the site of the lesion is always at the upper end of the shaft of the tibia. It occurs from $1\frac{1}{2}$ to $3\frac{3}{4}$ in. below the epiphysial line, depending on the age of the patient and the height of the individual. (For example, it occurred $3\frac{3}{4}$ in. below the epiphysial line in a patient aged 20 years.)

There is always callus, usually occurring on the inner aspect, but occasionally on the outer aspect. The early changes may very easily be overlooked unless the quality of the radiographs be faultless. In the lateral view the shadow of the fibula may obscure callus formation behind the tibia ; whilst in the antero-posterior view of the tibia, the upper end may be near the edge of the film and may be relatively under-exposed. Where there is uncertainty, further radiographs should be obtained seven to fourteen days later. If these fail at this time to reveal definite evidence, it may be presumed with safety that the condition was not present. The opposite leg should always be X-rayed for comparison.

In early cases there will be seen, at the site described, a tiny nick in the cortex of the tibia, either medially, laterally, or both. In the lateral view at the same level, there will be seen beyond the cortex posteriorly a tiny mass of callus. Later, extending forwards horizontally within the tibia from this callus will be seen an area of osteoporosis, sometimes with a fine irregular line of dense bone, the width of a hair, in the centre of this. In a mild case, in place of the area of osteoporosis, there will appear an ill-defined zone of osteosclerosis. In the more severe cases callus will begin to show in the antero-posterior view as well as in the lateral view. If the patient is rested the callus increases, the osteoporosis is replaced by dense bone, the callus becomes organized, and then during the ensuing months a gradual repair occurs. There is a tendency

for some local antero-posterior broadening to remain, due to organization of the callus posteriorly into a bony buttress similar to that seen after unreduced greenstick fracture of the lower end of the radius.

These radiological appearances may be seen within fourteen days of the onset of pain. I have never yet been able to demonstrate any significant changes in any other bone in any of these 14 patients.

PROGNOSIS AND TREATMENT

No case in this series, which has been treated solely by immobilization, has failed to progress to spontaneous cure.

In the 2 cases recorded by Weaver and Francisco no causative organism was found, and in their first case cultures taken from the bone-marrow proved to be negative. In no uncomplicated case that I have seen reported has pus been found. In 3 cases of which I have personal knowledge which were either trephined or opened up, no pus was found.

The indications are for absolute rest for several weeks, investigation for causes of abnormal stress and strain, and improving the general condition of the patient, with special emphasis on the calcium metabolism. As to the need for surgical interference—if the radiographs show, in the first week or two, that the bone is developing an area of sclerosis, it may be foretold with reasonable certainty that progress will be rapid and effective without any surgical intervention. Should the area of osteoporosis develop rapidly, then a case may be established for immediate surgical exploration. There is not yet enough material available on which to decide this question.

CASE REPORTS

In order to save space, only 3 cases will be illustrated and described here. No. 12 of my series, the most spectacular case, being bilateral, missed clinically, and proceeding to fracture of both tibia and fibula, is described here as *Case A*. No. 1 in the series, the case which brought the condition to my notice, showing typical advanced appearances, but which did not proceed to pathological fracture, will be called here *Case B*. No. 11 of the series is representative of the mild type of the lesion, and will be described here as *Case C*.

A B

FIG. 13.—*Case A*. Radiographs of left leg taken on the day of the accident, Nov. 1, 1940. A, Antero-posterior view; B, Lateral view.

Case A.—R. G., aged 21 years. Warehouseman. He is sparely built; a pale, freckled lad. He was conscripted on March 13, 1940, when he was 20 years and 4 months of age. He was passed fit, and was sent to Ireland for three months' army training, which period he described as "an easy time". In June, 1940, the Rifle Regiment, in which he was serving, began serious road training. On his first pace-march he had to drop out of the ranks because of pain in his *right* calf. Reporting sick, he was excused pace-marching for one month, and did light duties. Resuming training once more, he had to drop out again on his first pace-march, because of pain in his

left calf. This pain became steadily worse, but apparently nothing was done, and he was simply kept on light duties once more.

In October, 1940, when given three weeks' sick leave, he returned to his home, and went to the local emergency hospital complaining of severe pain in his left calf, but was told he could not be treated there. He was limping badly, and, going downhill on his way home, something happened suddenly and the patient fell. He was uncertain whether he slipped and his leg broke, or whether his left leg gave way and he then fell. He was promptly removed to the emergency hospital and X-rayed immediately. On Nov. 1, 1940, radiographs showed " fracture of the upper third of the left tibia and fibula. The fibular fracture is recent; the tibial fracture is not entirely;

A B

FIG. 14.—*Case A.* Radiographs of right leg taken 2 days after the accident to the left leg, and about 4 months from the onset of first symptoms—namely, pain in this left calf. A, Antero-posterior view; B, Lateral view.

obviously a re-fracture, with evidence to suggest that it has occurred through the site of previous ' fatigue fracture ' of the tibia. This opinion would be confirmed if radiographs of the right tibia reveal evidence of ' fatigue fracture ' there ".

My X-ray report on the *right* leg, on Nov. 3, 1940, was: " The right tibia shows indubitable evidence of the condition known as ' pseudo-fracture ' situated at the same level as that at which fracture has occurred in the left tibia. This finding confirms the diagnosis of pathological fracture of the left tibia, previously suggested."

Fig. 13 shows the appearances in the upper third of the *left* leg at the time of admission. It is obvious that the fibular fracture is uncomplicated, in contradistinction to the appearances at the tibial site. These appearances could not be due to recent fracture only. The callus visible posteriorly gives the clue to the condition. *Fig.* 14 shows the appearances at the upper end of the *right* leg two days later. To anyone who has seen the condition of ' pseudo-fracture ' previously, these appearances are unmistakable. They dovetail exactly with the history of pain occurring under severe strain four months previously. This leg having been given opportunity to recover, has made a typical attempt to reconstruct this weak area of the tibia, and is here seen well advanced towards cure. The left leg was put up in plaster-of-Paris and the patient kept at rest for many weeks, both legs being X-rayed from time to time.

There is an interesting series of films showing the left tibia and fibula gradually uniting soundly. Exactly six months after the fracture had occurred through the weakened area, firm bony union of both bones had occurred. The right tibia at this time still shows some broadening, and buttressing posteriorly, but the architecture has markedly approached normal. I think that at least six months were wasted here due to non-recognition of the cause of his pain at the first pace-march.

It would be very interesting to obtain data concerning the number of young recruits, particularly to Rifle Regiments, who fall out during their first pace-march training, and to know how many of these show eivdence of any pathological change at this site in the tibia, or of changes in the fibula.

My contention is that the cost of taking two small X-ray films in each case where a recruit complains of severe and persistent pain would well repay itself, should even a small percentage of those X-rayed prove to show this condition of fatigue fracture. In this particular case the cost of X-raying both legs would have been under ten shillings, whereas this man has already cost the nation four months' upkeep as a ' passenger ' under service conditions, and several months more under hospital conditions. He has been discharged from the Army—in my opinion quite unnecessarily—and he is still not a useful member of the community. I maintain that had this condition been recognized immediately, he would by now be quite capable of army service, although unsuited to rigorous marching.

Case B.—W. W., aged 18 years. Labourer.

He was apparently a healthy lad. He gave no history of injury, and complained only of pain of two weeks' duration at the upper end of the left tibia on weight-bearing. X-ray examination on Dec. 29, 1931, showed " slight erosion of the cortex on the medial and posterior aspects of the left tibia, about 3 in. below the knee-joint ". On Jan. 20, 1932, the radiographs showed that the erosion of the cortex had progressed, and a small node of periosteal new bone was seen postero-medially. On Feb. 17, 1932, the condition had progressed considerably, and there was then an area of osteoporosis extending across three-quarters of the antero-posterior width of the shaft. The bone had not, however, become fractured. Immediately above and below this area of rarefaction there was slight sclerosis, whilst in the middle there was to be seen a fine dense line. There was then periosteal new bone visible on the inner, outer, and posterior aspects of this area of the tibial shaft. No abnormality was detected in the opposite leg or in any other part of the bony skeleton; the boy was not ill, ran no temperature, and so long as he was at rest did not complain of pain.

On March 15, 1932, after further X-ray examination at which the condition was found to be still present, he was admitted to hospital. At exploratory operation, a cube of bone was removed from the inner

aspect of the tibia at the affected area, and was sent for histological examination. The pathological report did not throw any light on the aetiology. Recovery was uneventful, and the process of healing was demonstrated throughout a long series of radiographs. The boy was working when examined on July 23, 1934, and reported then that he had had no trouble since November, 1932. The total period of incapacity was less than eleven months. Inquiries were made in 1937, and again in 1940, and the patient is reported to be still working and leading a normal life without complaint

At the time this case was first X-rayed, no similar case had been seen. I was unable to find any radiologist who had seen this condition before, and my graphs showed typical early changes of fatigue fracture of the right tibia 3½ in. below the knee-joint. No pathological changes were detected in the left leg. Radiographs were taken at weekly intervals in order to study the progress of the condition, and to check up on the prognosis which one had been able to give in the light of 10 previous cases. The left tibia showed the typical changes and progress which one had come to expect in a case treated solely by rest in bed. Six and a half weeks later the patient had no pain on walking. New bone was then becoming organized.

This patient had had all the usual exanthemata. Five years previous to the first examination he had had an illness which was reported to be rheumatic fever. The patient is certain that there is no question of injury to this leg.

A B

FIG. 15.—*Case B.* Radiographs of left leg taken 9 weeks after onset of symptoms, showing maximum destructive changes. Repair began within the next month. A, Antero-posterior view; B, Lateral view.

diagnosis was: "a local osteitis of the tibia, of unknown origin".

Fig. 15 shows the typical appearances of fatigue fracture of more than usual severity, at the stage of greatest activity. It is the author's belief that at the stage shown here, two activities are being carried on side by side. The faulty bone-cells in the middle of the area are being rapidly removed, whilst on either side rebuilding and strengthening are proceeding. When the faulty bone has all been removed, new and adequate bone is laid down. Experience shows that unless rest is withheld, the natural processes will always adequately estimate the new strength and type of bone to be laid down in this area.

Case C.—J. P., aged 14½ years.

This boy was big and over-weight for his age. He was fat, had a double chin, and showed acne facialis. There was some blueness of both hands. On Sept. 20, 1940, he complained of pain in the right calf at times when he was walking. On Sept. 30, radio-

He made an uninterrupted recovery. Two months from the onset of symptoms he was able to resume normal activity. Nine months from the onset he is reported to be still symptomless, and his doctor reports that he can find no tenderness, swelling, or other clinical abnormality in this area or in the opposite leg.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis lies between:—

1. Simple Fracture.—This requires very great violence and seldom occurs without other damage, in this area. The total absence of history of injury in 9 cases out of 14, and the existence of the typical changes in both legs in 3 of the series, would seem to preclude this diagnosis.

2. Looser's Umbauzonen.—These occur chiefly in the ribs, the pelvis, or the neck of the femur. There will be evidence of advanced generalized bone disease. Fatigue fracture, in contradistinction, occurs without evidence of abnormality elsewhere in the bony skeleton. Moreover, *Umbauzonen* remain static, whereas tibial fatigue fracture changes rapidly from week to week, always progressing to cure.

3. Osteomyelitis. — Clinical differentiation from acute osteomyelitis should present little difficulty, for, of the classical symptoms, only pain is common to both, and even this in fatigue fracture disappears on rest. Clinical distinction between chronic osteomyelitis and fatigue fracture would be difficult. Radiologically the difference is striking: the bony changes extend transversely in fatigue fracture, whereas longitudinal extension, less sharp delimitation of the intraosseous changes, and elevation of the periosteum are pathognomonic of osteomyelitis.

In *none* of the 3 cases in my series in which surgical exploration was performed was pus to be found.

4. Sarcoma.—The differential diagnosis is entirely radiological. An early or mild case of fatigue fracture might be mistaken for an unusually early sarcoma, were one not fully alive to the characteristic appearances seen in fatigue fracture —the clean-cut 'nick' in the cortex, the tiny node of callus, and the entire absence of any bone destruction in the vicinity. Later the characteristic narrow transverse zone of bony change, with the relatively broad buttress of regular callus, is easily distinguishable from any malignant process, and within three weeks of the first radiograph the diagnosis will be beyond dispute radiologically.

DISCUSSION

It will be noted that until recently the condition has been investigated almost entirely from a radiological viewpoint. Obviously there remains much research to be carried out in the clinical, biochemical, orthopædic, and pathological fields. There is a considerable amount of evidence to show that this area of the tibia is subject to a great deal of strain. It will be observed, in the routine examination of the legs in all types of cases sent for radiological investigation, that it is quite frequent to find either sclerosis of the cortex or some buttressing in this area in adults.

In the antero-posterior radiograph there is often to be seen a buttress on the outer aspect, and a similar buttress on the inner aspect at a slightly lower level, or occasionally at the same level. When they are seen it will frequently be observed that the shape of the bone and the architectural arrangement of the trabeculæ suggest that the body-weight is not being normally transmitted. I am certain that if research could be adequately made, combining orthopædic examination and radiographs of the spine, pelvis, and lower limbs, this point could be readily established. The frequency with which these bony buttresses are seen, it is suggested, lends colour to the belief that this represents one form of a normal strengthening process occurring in children and adolescents.

It is my belief that the condition is a fatigue fracture due either to faulty crystal structure of bone at this area of maximum strain, or to abnormal strain at a site already subject to considerable stress. Or it may, of course, be due to a combination of both. It is essentially a fatigue dystrophy such as has been described by Professor C. Henschen of Basle.[6]. Its similarity to march fracture of the metatarsals, both in radiological appearance and in its rate and method of healing, is striking. This similarity has been widely commented upon, being mentioned in most of the publications which I have seen, and it is almost invariably remarked upon by those to whom I have shown my series of radiographs.

It is definitely not due to infection; it is not bone tumour; it bears no relationship to Looser's *Umbauzonen*; and there is no evidence that it is due to syphilis or to tuberculosis.

SUMMARY

1. Fourteen cases of fatigue fracture of the tibia are recorded.

2. Theories regarding the probable nature of the condition are advanced.

3. The importance of early recognition of the disease in wartime is stressed. It is held to be a fatigue dystrophy and not of infective origin, nor due to direct violence.

4. Treatment is discussed and the results of immediate immobilization in plaster are recorded.

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THE ILEO-GASTRIC SYNDROME*

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A SYNDROME is a set of symptoms or signs which occur together, or the sum of signs of any morbid state. In 1932 Gaither,[1] in an article entitled "Abdominal Syndromes of Exceptional Interest, their Underlying Pathology", refers to this and other syndromes, but not under this title. No apology is made for it, because it is believed that the more we realize the close association between the ileocæcal region and the stomach and liver the more quickly we shall reach a position in which to classify, diagnose, and treat the pathological conditions concerned, by medical or surgical means.

It would be right to say that during the last twenty-five years of his active surgical career Moynihan never opened an abdomen without closely observing and recording (almost always with his own hand) the condition of the appendix, which he invariably removed; from a large number of these records he collected the material on which he based his address "The Pathology of the Living", published in 1907,[2] and "Appendix Dyspepsia" in 1910.[3]

It is on a correlation of at least a thousand of his drawings and observations, together with even more of those of my surgical colleagues, that this thesis is based. Doubtless Moynihan would agree with the choice of a title even if he did not approve of the manner of presentation.

The events which concern the ileo-gastric syndrome fall into two parts :—

I. Those arising in the gastric area (the upper pole), producing phenomena in the ileocæcal region, i.e., the 'gastro-ileal reflex'. A normal non-pathological sequence, and a true nerve reflex.

II. Those arising in the ileo-cæcal region (the lower pole), giving rise to symptoms and signs in the 'gastric' area, and probably of pathological origin alone, the 'ileo-gastric syndrome' proper.

I. THE GASTRO-ILEAL REFLEX

In the course of the Huxley Lecture in 1904[4] Sir William Macewen showed that the cæcum and appendix were concerned together in intestinal digestion. He based the truth of his contention on observations on monogastric herbivora (horse, kangaroo, and rabbit), in which the cæcum is enormously developed, being much larger than the stomach and abundantly supplied with gland follicles and large lymphatics. He maintained that "the cæcum is thus a compensatory organ to the stomach" dealing with the digestion of cellulose. He quotes a case of a man whose anterior cæcal wall had been removed by an explosion, revealing the ileocæcal valve and the appendicular orifice (valve of Gerlach). On this patient he made very careful observations, the chief of which was to show the existence of the gastro-ileal reflex. When fluids or solids entered the stomach the contents of the lower ileum poured through the ileocæcal valve to be met by fluid exuding from the appendix and cæcum. He regarded the appendical and cæcal fluid as, in some way unknown, influencing the process of digestion. He suggested that subacute or chronic disease of the appendix might cause disturbances of digestion.

In more recent times the gastro-ileal reflex has proved of invaluable assistance in X-ray diagnosis where investigation of the ileocæcal region is required. As is well known, and described by Hurst (1909) and others, there is a perfectly natural holding up of the contents of the ileum at the ileocæcal valve—Hurst regards it as a valuable part of intestinal digestion. At the end of about two hours after ingestion food reaches the valve, but it is not until another two hours it enters the cæcum. If, however, a further meal be given, the contents pass at once to the cæcum and the X-ray diagnostician is enabled to carry on his further investigation.

In the opinion of Harnett and Galstown.[5] of Calcutta, the gastro-ileal reflex is found in 85 per cent of all cases.

Bruce Maclean[6] considers that a delay in filling the cæcum indicates chronic appendicitis. It may be that the recognition of this reflex underlies the generally accepted starvation treatment of acute appendicitis.

II. THE ILEO-GASTRIC SYNDROME

The ileo-gastric syndrome is in some measure the opposite of the gastro-ileal reflex, functioning from below upwards but probably depending on pathological rather than nervous influences. We have to consider two ends of the chain of transmission: (*a*) pathological conditions arising in the ileocæcal angle, the primary focus; and (*b*) the pyloric end of the stomach, the duodenum, the liver, and gall-bladder, the sites of the end-results.

In the ileocæcal angle three chronic pathological conditions will be shortly described: (1) Chronic appendicitis; (2) Simple chronic ileocæcal adenitis; (3) Chronic tuberculous adenitis.

It will be understood that such symptomatology and physical signs as may be allotted to each

* Being the substance of the Moynihan Lecture, delivered Nov. 4, 1941, at the Royal College of Surgeons, London.

of the three diseases have been elucidated by consideration of a very large number of records and have been confirmed by subsequent operation.

Nothing will be said of the grosser pathological conditions like tuberculosis or malignant disease of the bowel in the ileocæcal angle, nor of Crohn's disease, though it will be necessary to mention ileitis as it affects the lowest segment of the ileum; in these diseases operation is demanded by reason of the local condition and not by a concurrence of symptoms elsewhere.

1. Chronic Appendicitis or Appendix Dyspepsia (*Fig.* 16).—It is immaterial whether or not Moynihan in his paper of 1910, "Remarks on Appendix Dyspepsia",[3] was the first to describe this now almost universally accepted

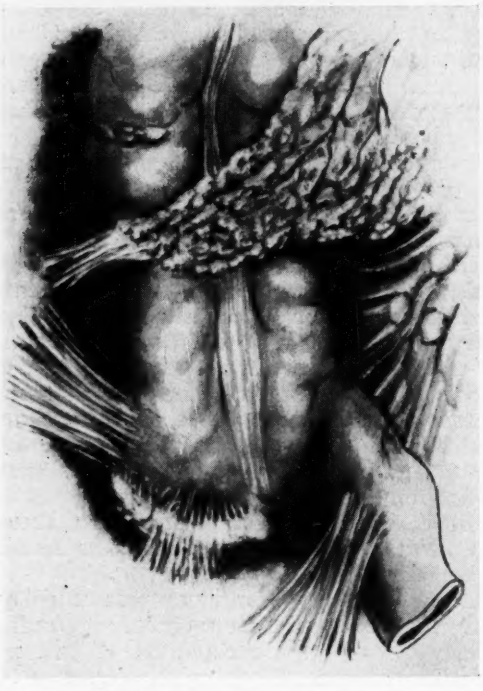

FIG. 16.—Showing the common adhesions round the cæcum following repeated attacks of appendicitis.

disease; he was in any case the first to describe it and to make it of real interest to surgeons, and to him must go the chief honour of its exposition.

Ewald, of Berlin, claimed that he described appendix dyspepsia as "Appendicitis Larvata" in 1899; he said: "the physician thinks of everything except the appendix". Herbert Paterson claimed that he and Dr. Soltau Fenwick described it in 1910 under the title of "Appendicular Gastralgia", spoke of the gastric hypersecretion accompanying it, and said that the Mayos had taught it for years. Blair Bell wrote to the *British Medical Journal* in 1910 and indicated that he described the pathology, symptomatology, diagnosis, and treatment of indigestion associated with chronic appendicular disease six years before.

Moynihan's article was followed by a spate of correspondence, varying in kind from Bowlby's outspoken letter of Feb. 5, 1910, in which he says, "I think that one result of Mr. Moynihan's paper will be that many 'dyspeptic' people will undergo operations for removal of the appendix and that the great majority of them will be none the better", to Clifford Allbutt's scholarly letter of Feb. 12, "I am disposed to anticipate partly by analogue of other such vicious circles, that a fine discrimination of symptomatic series and an accumulation of data will prove that latent disease of the appendix may not infrequently, in persons of low resistance, set up reactions of a dyspeptic and 'neurotic' kind". Since Moynihan's paper a great deal of observation and research have been done, and it is only possible in this article to skim over the chief features of the diagnosis.

In the General Infirmary at Leeds nearly 200 cases a year are labelled chronic appendicitis, but investigation of the records shows that only 50 to 60 are cases of 'appendix dyspepsia'; a further 50 cases a year are definitely described under this title and operated upon as such; there are, therefore, roughly 100 cases a year for consideration.

SYMPTOMS.—The disease occurs in young adults and in people up to 50 years of age; some consider it a familial disease. There is equality in incidence in the two sexes; sometimes there is a history of acute appendicitis years ago, more often none at all; many look very well, some appear obviously dyspeptic. The striking point of the symptomatology is 'variability' in frequency and in kind of pain. The epigastric discomfort, sometimes a real pain, is truly epigastric, that is, *above* the umbilicus (the site of Morley's true splanchnic pain in acute appendicitis and other acute abdominal conditions is at and around the umbilicus itself); it follows or precedes food, usually the former; it may come by day, or rarely, by night; it is in every particular irregular. One striking feature is gastric flatulency, like that in chronic cholecystitis (which may indeed also be present); sometimes it is so obvious that refined people are afraid to take their meals in company. Days, weeks, or even a year may pass without symptoms. As in duodenal ulcer the trouble is worse in spring and autumn. On the one hand, the symptoms mimic a duodenal, on the other, a gastric ulcer. Exercise and mental worry accentuate the trouble. Occasional local appendicular pain may occur. Medical treatment is usually ineffective.

Hæmatemesis is rare; in our series it occurred in only 4 per cent. This striking symptom has been mentioned by Moynihan. It is, of course, well known that hæmatemesis occurs with great frequency in acute appendicitis—almost all cases have an early 'black vomit' which contains blood. Hutchinson[7] says "twenty-one of twenty-four cases of fatal hæmatemesis were due to appendicitis with septic complications, local peritonitis or abscess".

It is appreciated that such an account of symptoms is of little value, unless one appreciates that their very indefinition and variation constitute a strong lead to diagnosis; but two methods of investigation, the Rehfuss test and X-ray examination, are of more definite utility:—

THE REHFUSS TEST.—Bennett and Ryle, who examined the gastric secretion of 100 students who never suffered from any form of indigestion, found that 80 per cent were within normal limits, 10 per cent had a constitutional hyperchlorhydria, and 10 per cent hypochlorhydria or achlorhydria. In appendix dyspepsia examined by the Rehfuss test pyloric spasm is found by inference in rather less than 50 per cent of cases definitely proved by it plays a big part in digestion, and its variation in quantity or quality will one day be found to have a bearing on the causation or persistency of peptic ulcer. Hermon Taylor[8] considers that gastroscopy may show physical signs of the presence of pyloric spasm. If the Rehfuss test is 'positive', it is to be regarded as an indication of pyloric spasm which *may* be produced by chronic appendicitis, simple or tuberculous ileocæcal adenitis, or other pathological conditions in that area.

X-RAY EXAMINATION.—

a. Gastric.—Evidence of pylorospasm is by no means easy to obtain, but is seen in 40 per cent of cases afterwards proved by operation. It is chiefly shown in delayed gastric evacuation

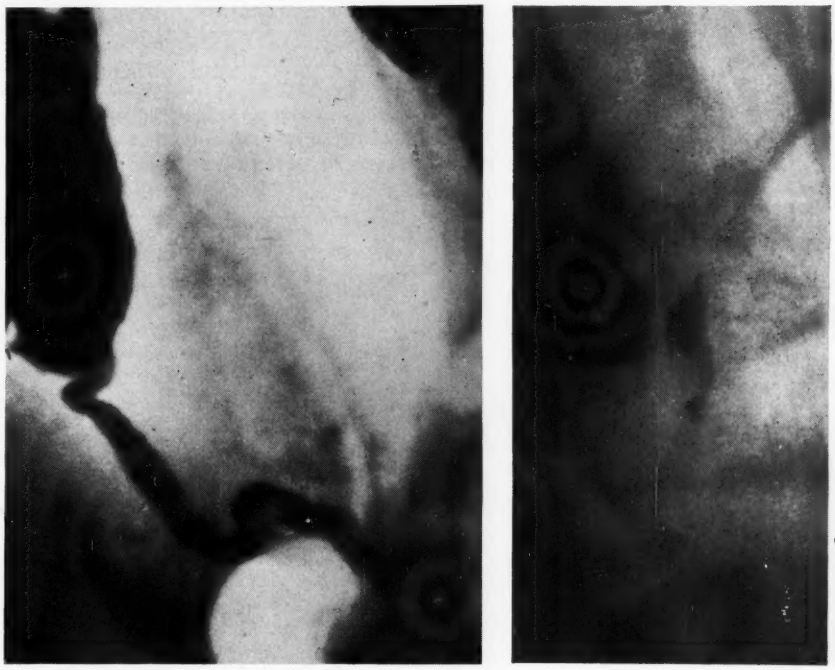

FIG. 17.—Kinked appendix near base—5 days' delay. The right figure shows the condition 3 days after the cæcum had emptied. (*Marxer.*)

subsequent operation and symptomatic recovery. Spasm and probably congestion result in retention of gastric juices, moderate hyperacidity, rarely achlorhydria, and a very striking diminution or absence of bile three hours after food when it should normally be present in quantity. Normal regurgitation of bile has never received the recognition it deserves; it is said by those familiar with the use of the gastroscope that there is far more than was at one time appreciated, but the presence of a gastroscope, like the presence of a Rehfuss tube, may easily modify its appearance both in time and quantity; in the same way the degree of acidity may be influenced. In every case of true pyloric spasm, whatever its cause, there should be a great reduction or a complete absence of bile. Modern books on physiology say nothing or very little of normal bile regurgitation; of its importance there can be little doubt; with increased peristalsis, but may be accompanied by a definitely visible spasm, sometimes mid-gastric, according to Hurst sometimes reversed peristalsis. Wilkie[9] described the X-ray appearances in appendix dyspepsia as (1) slight delay in gastric emptying, (2) a spastic duodenal cap, and (3) stasis in lower ileal coils. Many others have reached similar conclusions.

b. Ileocæcal.—

i. *Appendix*: A delay in filling of cæcum and appendix due to spasm of the ileocæcal valve, i.e., a delayed 'gastro-ileal reflex', delayed emptying or stasis, signs of *abnormal* filling due to adhesions or kinks, presence of fæcoliths, indications on manipulation of fixation of the appendix, cæcum, or lower ileum (Lane's ileal kink), local tenderness under manual examination, often with pain referred to the umbilical or epigastric areas (*Figs.* 17–19).

ii. *Ileal Stasis*: Confirmed by many observers, and by some regarded as the most significant sign of chronic appendicitis, certainly

FIG. 18.—Narrowing of distal appendix from fibrous tip. (*Marxer.*)

due to spasm of the ileocæcal valve and not to a Lane's ileal kink, which indeed would cause any 'delay' to be permanent.

FIG. 19.—Chronic anchored appendix in erect posture—cæcum dependent from it. (*Marxer.*)

PHYSICAL SIGNS.—There may be appendicular tenderness. Bastedo says that local tenderness over the ileocæcal region after colonic inflation is common when there are adhesions round the appendix. Rovsing's sign consists of the demonstration of pain over the appendical region when pressure is exerted on a similar point on the left side. There may be evidence of gas accumulation in the cæcum; there may be (Aaron's sign) a referred umbilical or epigastric pain on pressure over the appendix—these were more or less definitely observed in 30 per cent of cases proved by operation. As a rule, there is no true *epigastric* tenderness unless there happens to be an exacerbation of an attack in progress, and then it may be due to either pylorospasm or local subpyloric adenitis, for at operation enlargement of the ileocæcal group of glands is usually found; such enlargement is not a marked feature, the glands are not red, nor do they contain any infective organism.

In appendix dyspepsia, therefore, the symptoms are epigastric, the signs both epigastric and ileocæcal.

2. Simple Chronic Ileocæcal Adenitis (*Fig.* 20).—All surgeons must remember cases where an operation for subacute or acute appendicitis revealed no such condition, but a mass of red

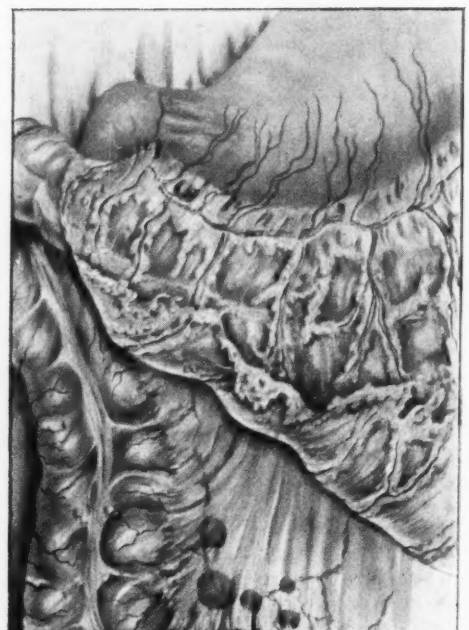

FIG. 20.—Simple chronic ileocæcal adenitis.

glands in the ileocæcal angle; in some cases they may have observed an increased reddening and peristalsis in the lower ileum—this condition occurs in young children and in boys about the age of puberty; they have a history of bilious

attacks and sometimes of acidosis. One has operated on many such cases. They all show a rise of temperature up to 100° or 103° F., localized ileocæcal pain and tenderness from the beginning, vomiting is frequent, and sometimes free fluid is found in the abdomen. In many cases the attack is associated with tonsillitis.

My first recorded case is as follows :—

A. B., a boy aged 14, seen with Dr. Walter Longley, was seized with sudden pain in the right iliac fossa on Feb. 2, 1934. No vomiting, evening temperature 103°, pain was definitely localized but radiated over the whole abdomen. Tenderness and rigidity well marked over site of pain. Pulse 110°, tongue clean. History of many less severe but similar attacks and sometimes of associated acidosis with vomiting. Differential diagnosis from acute appendicitis could not be made, but the clean tongue, the high temperature, and the general appearance of the boy made appendicitis unlikely.

Right paramedial incision—appendix, very red and long, removed, many red glands in the ileocæcal angle ; one of them was removed and found to be inflamed and to contain the hæmolytic streptococcus ; the last six inches of the ileum were intensely red and thickened—there was no lymph and no free fluid. Abdomen was closed without drainage and recovery was uneventful. There has been no return of trouble. Investigation of appendix showed only congestion of peritoneal and subperitoneal coats.

But the *chronic* variety is commoner and occurs in young adults. Fifteen cases have been considered ; it is regretted that investigation by the Rehfuss test and X-rays was so rarely carried out. These cases are distinguished by two definite features—a rise of temperature during exacerbation of the disease, and a local tenderness. The glands are sometimes definitely in the mesentery of the lower ileum and sometimes in the ileocæcal angle proper, usually in both. Occasionally at operation one finds spasm in the lower ileum, and there is no hesitation in diagnosing a subacute ileitis. On microscopical examination, the glands show the usual signs of inflammation, and sometimes (in four cases) revealed the hæmolytic streptococcus, which was also found by Deaver. In addition to a rise of evening temperature and the presence of a local tenderness, there is an 'indigestion' like that of appendix dyspepsia, but less severe. The Rehfuss test, of which only six can be quoted, is similar to that of appendix dyspepsia. The X-ray in five cases out of nine examined showed intermitting pyloric spasm with similar features to those of appendix dyspepsia ; X-ray examination of the ileocæcal region showed both ileal and ileocæcal spasm : in four cases there were attacks of acidosis with abdominal pain. In no case was a correct diagnosis made before operation.

It is almost impossible to imagine that operation in itself can do any good for this condition ; the trouble is to differentiate it from subacute appendicitis ; so far it has been impossible to do so. Operation is inadvisable if it can be avoided, but so far radiologists are not always prepared to swear to the good health of an appendix, in fact, few appendices examined seem to possess it ; it would appear inevitable in view of the local pain and tenderness that operation (with removal of the appendix) is essential. Such cases do in fact recover quickly after operation, but it is by no means certain that the appendix is the cause of the adenitis though it may appear redder than is normal. Recovery is probably due to the dietetic restrictions, bowel regulation, and enforced rest which follows every operation on the abdomen. It is difficult to get away from the probability that this condition is wholly due to a local infection—ileum, appendix, or cæcum. If ileal it may be the result of some form of intestinal or generalized poisoning ; if appendico-cæcal it may follow a low-grade infection of the appendix. Simple ileocæcal adenitis is much commoner than we know, and surgeons who look at this area in the course of all abdominal operations will find it oftener than they may think. Here is a field for much wider investigation, particularly in the possible association of chronic infection in other areas, especially the tonsils and pharynx.

3. Chronic Tuberculous Adenitis.—This is a condition of which the author has a large experience ; his description of it appeared in 1926.[10] At that time there was no evidence available of the results of the Rehfuss test or of gastric X-rays in these cases. The writer had under his own care during the last ten years an average of 20 cases a year ; the General Infirmary at Leeds now has a yearly average of 50. The disease in this form is of young adults ; it represents the triumph of resisting power over tubercle, and it is therefore not surprising to find it commoner in private than in infirmary practice. The symptoms are not immediately due to the tubercle bacillus but to the mass of lime salts left behind after the conflict with it ; a local irritation is set up and often a definite local peritonitis, sometimes with the formation of fibrinous flagellæ attached to exposed points of the calcified gland. As might be expected, massage or violent exercise, particularly vigorous extension of the right hip (such as occurs in serving at tennis) commonly causes an exacerbation. But in the quiet intervals other signs than those of *local* irritation may be found : (1) spasm of the ileocæcal valve ; (2) localized spasm of the ileum exactly opposite a calcified gland which happens to be near the ileal mesenteric border : (3) pylorospasm with increased peristalsis ; hypersecretion, hyperacidity, and delay or absence of bile in the stomach. Numbers 1 and 3 of these signs were shown by X rays as well as by the Rehfuss test in approximately 30 per cent of the 24 cases recently examined.

Radiographs of the ileocæcal region usually show the typical picture (*Fig.* 21).

The local symptoms and signs are, as one would expect, localized pain and localized

tenderness. It is rare to find any rise of temperature; if any is present it may indicate an added infection or that tuberculous disease is still active. Examination of glands excised have shown active tubercle bacilli only rarely and other infections more rarely. It may, therefore, be truly said that the local symptoms and signs are those of trauma. Sometimes a chain of calcified glands is found reaching from the ileocæcal angle to the portal fissure—they have been mistaken, on palpation, for stones in the common bile-duct; occasionally the glands are so extensive that operations for their removal cannot be contemplated.

The three conditions I have so superficially described can be differentiated by X-ray examination *of the ileocæcal angle* :—

1.—Appendix dyspepsia: definite localized X-ray evidence in the appendix and spasm of ileocæcal valve.

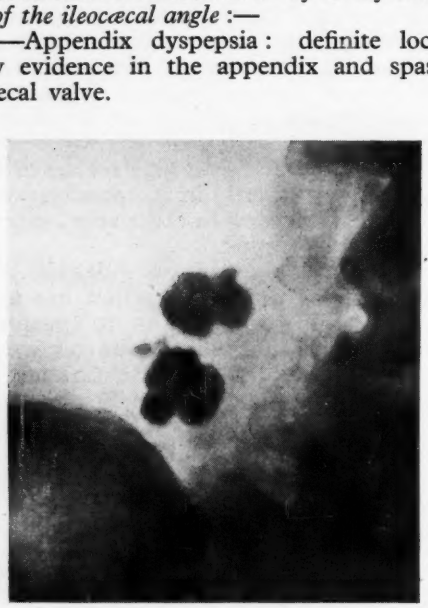

FIG. 21.—Typical calcified ileocæcal glands. (*Rowden.*)

2. Simple chronic adenitis: spasm of lower ileum and ileocæcal valve.

3. Chronic tuberculous adenitis: X-rays of glands, spasm of lower ileum and of ileocæcal valve.

In all three, epigastric signs of pyloric spasm with hypersecretion, hyperacidity, and delayed or non-appearance of bile in the stomach may be the same.

The upper end of the syndrome in all three diseases presents to the surgeon at operation in any average case the three visible stigmata which Moynihan associated with appendix dyspepsia alone: (1) pyloric spasm; (2) pyloric congestion visible only in the quiescent non-spasmodic period; and (3) enlargement of a lymphatic gland on the greater curvature of the stomach, usually 1 in. to the left of the pylorus and frequently hidden in fat (*see Fig.* 20). Writing in 1904, Moynihan said :—

On several occasions during the last few years I have watched the stomach intently for several minutes, and have seen the onset, the acme, and the gradual relaxation of a spasmodic muscular contraction in its walls. Quite gradually the stomach narrows, and the wall becomes thicker and almost white in colour; when taken between the fingers the contracted area feels like a solid tumour. The spasm may be so marked as to prevent a finger being invaginated through the segment affected. The appearance presented is very striking. I have seen it in the body of the stomach and at the pylorus, but never at the fundus. As slowly as it comes on the spasm quietly relaxes, and the stomach assumes its usual form.

The three signs are easily seen if carefully looked for, and had been noted long before X-ray diagnosis of gastric and duodenal ulcer was anything like so definite as it is to-day in the many cases where a high incision had been made to deal with an ulcer and where no ulcer was found; it was Moynihan's practice to say: "here are the three stigmata, we are dealing with a bad appendix and not with an ulcer". But the same three stigmata may arise in ileocæcal glandular disease, and Moynihan was probably not correct in always attributing the trouble to the appendix alone.

THEORIES OF CAUSATION OF THE SYNDROME

The manner in which the syndrome works, that is, the method of transmission of the impulse from below upwards, remains doubtful, but there are at least two theories.

1. The Lymphatic Theory.—This has been propounded by Ginsburg, by Descomps and Turnesco, and in the Arris and Gale Lecture of 1923.[11] Remembering the teaching of Lane, the high infectivity of the contents of the lowest part of the ileum, and realizing that simple and tuberculous adenitis may be set up by it, remembering, too, the frequency of disease in the appendix resulting largely from retention of its contents, and not forgetting the possibility of infection from the cæcum, there is a fruitful field for infection in the ileocæcal angle, probably the most fruitful in the whole abdomen. The author[12] writes :—

Let us assume that we start with perfectly healthy lymph-vessels and glands. For some reason or other there comes pouring into the mesenteric group of glands a stream of infected lymph; the result is bound to be a lymphangitis and a lymphadenitis, spreading upwards from node to node with a gradually decreasing effect as it nears the last line of gland sentinels before reaching the blood-stream. Assume the flow still goes on over a period of months or years; it is not too much to suggest that chronic obstructive lymphangitis and lymphadenitis develop, most markedly nearest the origin of the infection, but gradually, involving the whole lymph-shed. The result of this change would be obstruction in the path of the normal flow. Gradually the whole mechanism is thrown out of gear, and we have everything present to give rise to aberrant and retrograde lymph-flow. Now, the infected lymph, failing to make its usual exit, seeks new avenues of escape in

all directions; misses whole groups of glands which under ordinary conditions would check and filter it, ebbs and flows to and fro until the glands around the superior mesenteric artery are reached and partly obstructed. Deprived of easy access through them to the lumbar glands, part of the lymph flows onwards over the head of the pancreas and enters the glands on the concavity of the duodenum, and in process of time bathes even the duodenal wall itself, escaping finally to the cœliac glands by normal or possibly aberrant paths (*Fig.* 22).

There can be little doubt that under pathological conditions infected lymph flows up on the right side of the abdomen in front of, through, and behind, the head of the pancreas to reach the stomach, duodenum, and gall-bladder, and probably the liver itself. The immediate effects of such a flow are subpyloric adenitis, pyloric and gastric congestion and spasm, increased peristalsis, hypersecretion and hyperacidity in the stomach, delay or absence of regurgitant bile, erosion of Peyer's patches in stomach and duodenum sometimes causing hæmatemesis, followed eventually by gastric or duodenal ulcer. A further effect may well be chronic cholecystitis, the formation of gall-stones with hepatitis and its diverse effects.

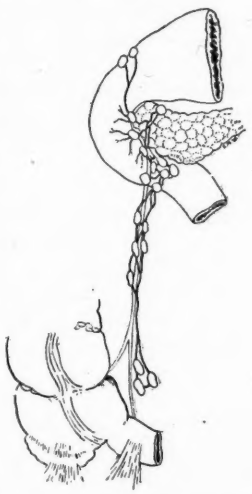

FIG. 22.—Diagram showing lymph-flow from ileocæcal angle to duodenum, stomach, and pancreas under pathological conditions.

2. The Nervous Theory. — Deaver[13] is of opinion that the irritation produced at the lower end of the chain passes to the upper end through the sympathetic nerves. Impulses set up in the appendix pass along the line of the ileocolic artery through the superior sympathetic plexus and set up 'epigastric pain, increased peristalsis, and pyloric spasm. Moynihan quotes W. B. Cannon and F. T. Murphy, who, in a most interesting article, describes a series of experiments carried out upon cats. In the cat, ordinarily, food begins to leave the stomach within ten minutes of ingestion, as is shown by examination with the X-ray screen after an opaque food (bismuth and potato) has been given. After a high intestinal operation—an end-to-end anastomosis, for example—the food begins to leave the stomach only after five or six hours. The delay is not due to etherization or to division of the abdominal wall; it is dependent upon an alteration in the action of the pylorus. After the operation peristaltic waves are seen to be coursing regularly over the stomach to the pylorus, precisely as they do in health. The difference is in the fact that the pylorus refuses to relax. Cannon and Murphy then show that the protective mechanism of the pylorus is beautifully proportioned to the needs of the intestine, for it is exerted during almost precisely that period (six hours) which is necessary for the repair of the intestinal injury. The function of the pylorus would, then, appear to be this: that, being controlled by stimuli aroused both on the proximal and distal sides, the distal stimulus, undoubtedly the more important, in altered conditions of the intestine, may be so insistent that relaxation is prevented and a firmer action is excited. The pylorus, that is to say, acts as a guard to the bowel distal to it, and it is probably true to say that its protective control extends to the whole of the mid-gut.

Sir William Macewen says:—

> The appendix is regarded as a mere diverticulum of the cæcum, and yet its vascular and nervous supply pertain more to that of the small intestine than that of the colon. The appendix is supplied by the terminal branches of the same group of nerves as supply the small intestine—the superior mesenteric plexus of the sympathetic. Reflex action may be easily set going in the appendix by stimuli from the small intestine, and each part may react on the other.
>
> When it is recollected that the circular muscles of the cæcum are continuous with those of the appendix, and that the longitudinal cæcal bands end themselves on the appendix, it will be understood how easily the nervous apparatus of the appendix may initiate the larger movements of the cæcum by first inducing movements in the appendix, and how inhibition of these movements may cause cæcal disturbance. The same agency, by control of the vascular supply, will regulate the exudation from the appendix, and that in accordance with the impulse received from the small intestine. It is known that the mind has an influence on the small intestine through the vagi, sometimes exciting peristalsis and sometimes arresting it. The appendix is subjected to the same mental effects.

The close nervous and lymphatic intercommunication between all abdominal organs makes possible the formulation of alternative nervous paths; the closer embryological association between the ileocæcal area, the pancreas, liver, and stomach suggest that the nervous theory has basic reasons which cannot be ignored. The lymphatic glandular enlargement and the end-results, that is, peptic ulcer, cholecystitis, and hepatitis, seem only likely to occur as a result of infection. On the other hand, in the early stage of local irritation in all three there is much that could be said for the nervous theory. May not each contribute a factor?

Attention has been drawn to a remarkable book by Elemer Forrai,[14] a Hungarian. It contains a foreword by Hurst, who epitomizes to a large extent the contents of the book. Hurst says:—

> There has in the past been too much tendency to regard the many disorders of the abdominal viscera as isolated phenomena. Physiological, pathological, and clinical investigations show, however, that there

is continuous interplay between the different organs, and that derangement of one is likely to have far-reaching effects on others. It is for this reason, among others, that the removal of a pathological appendix, except for acute attacks, often leads to little or no improvement in the patient's condition, so that the once popular diagnosis of chronic appendicitis or appendicular dyspepsia is now made comparatively rarely; some physicians even doubt whether such a condition really exists. When, however, it is realized that appendicular disease is often associated with achlorhydric gastritis, gastric and duodenal ulcer, cholecystitis, and an abnormally acting cæcum and ascending colon, the pathological appendix can be studied in a proper perspective. The same applies to operations for chronic cholecystitis as distinct from acute suppurative cholecystitis and gall-stones. The fact that an appendicectomy scar is one of the commonest physical signs of chronic cholecystitis is not so much an indication that a wrong diagnosis has been made in the first instance as that the case has been judged from a too narrow point of view.

The author has not lost sight of the facts of interplay between all abdominal organs and of the effects of disease of one of them upon the others. It is not pretended, however, that more than those described have been studied; it is not doubted there is a wide field for further research. No claim is made to have elucidated anything more than the association of at least three pathological ileocæcal conditions, which in correlation with their epigastric symptoms and signs seem worthy of consideration as a definite syndrome. The writer cannot agree with Hurst that the "once popular diagnosis of chronic appendicitis or appendicular dyspepsia is now made comparatively rarely"; it is, in fact, made almost as often as before and certainly with much greater exactitude. It may be true that "some physicians even doubt whether such a condition really exists"; it is remembered that much the same was said of duodenal ulcer, that gastric ulcer was a common disease and duodenal ulcer rare, and that if duodenal ulcer really occurred it could not be diagnosed before operation. However, it is not possible at the moment to diagnose appendix dyspepsia in the absence of local signs or symptoms. Hurst's contention that "an appendix scar is one of the commonest signs of chronic cholecystitis" would doubtless have been answered by Moynihan—the trouble in those cases has been not that the appendix was removed, but that it was not removed soon enough. There are pathological conditions in the ileocæcal region other than appendicular which can and do give rise to symptoms of dyspepsia—but the appendix is far and away the commonest offender. Might it be suggested that the condition now called "appendix dyspepsia" be called "ileocæcal dyspepsia"? Such a title ought not necessarily to indicate that the treatment is by removal of the appendix or indeed removal of anything at all: it may be entirely medical, for example, in simple ileocæcal adenitis. It is not assumed that the three conditions described in the ileocæcal angle are the only ones capable of producing epigastric symptoms and signs: it is realized, for example, that pathological conditions in the cæcum and colon may do the same, but they do so more rarely, and probably secondarily to those described.

Abdominal surgery, like medicine, may with advantage seek for its further advance a still deeper and wider knowledge of symptoms and signs correlated by surgical observations at operation—there is far too much operating in the hope that rather by chance than be reason the patient may be cured; this is, of course, particularly true of the appendix. In all good time the elucidation of the symptomatology in this area will be made clear and many unnecessary operations avoided. It may be that there is one central focus from which emanate all the changes described—it is even possible that such a focus may even be, as Forrai thinks, in the liver; this would indeed be strange, but even if it be true would not the knowledge of the existence of the gastro-ileal reflex make their transmission even from above downwards possible?

It is very much more likely that the syndrome is bipolar in action as well as in effect, but under pathological conditions the impulse seems to be much more active from the lower pole than from the upper.

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ECTOPIA VESICÆ COMPLICATED BY ADENOCARCINOMA, WITH A REVIEW OF THE LITERATURE

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ECTOPIA vesicæ is an uncommon congenital deformity. According to Neudorfer, it occurs once in every 50,000 births. W. E. Lower, of Cleveland, estimates that 2000 occur in the U.S.A. each year; some 650 might be expected to occur annually in Great Britain. Numbers of these tend to gravitate to those surgical centres which have built up a reputation in the treatment of the condition, but elsewhere the anomaly is rarely seen. According to Mayo and Hendricks, 50 per cent of all persons afflicted with exstrophy are dead by their tenth year, and 66·67 per cent are dead by their twentieth year. Buerger stated that out of 74 not operated on, only 23 passed the twentieth year of life. It is estimated that some 10 per cent reach maturity, and of this number a few develop carcinoma of the exposed bladder. The attitude which still lingers, that nothing can be done for these unfortunate babies, prevents the parents of many of them from seeking treatment and leads to despondency and neglect. That this is the main cause of the high mortality in the early years, is the opinion of Grey Turner, who stresses that the mothers should be seen at an early stage and encouraged to give the same devoted attention to these children as they would to their normal offspring. In a few cases, dilatation of the upper urinary tract is a concomitant feature, and infection with the resultant pyelonephrosis soon intervenes. This may show itself in the failure of the child to thrive in spite of receiving the best attention. In most cases, however, the upper urinary tract is normal, and can resist even gross infection of the bladder area, as is noted when this has been the site of malignant ulceration over a long period. Social outcasts from birth, in childhood they are denied education, and in adolescence they seek isolation in outdoor occupations, often arduous in nature, but for which they are otherwise physically fit. They often hide their deformity until some unbearable complication forces them to seek treatment.

It is a great rarity to see a case of ectopia vesicæ complicated by carcinoma. Since 1895, when Bergenhem reported the first case, 25 others have been added by authors in various parts of the world (*see Table I*). One only is to be found in British literature, described by Percy Sargent in 1910. The two cases described below make a total of 28. The most interesting point, and one which merits further discussion, is that in 23 out of 25 of the cases which were subjected to microscopical examination, the structure of the tumour was found to resemble closely that of an adenocarcinoma of the rectum. One's interest in the matter was aroused by the findings in the following case:—

Case 1.—M. T., male, aged 48, farmworker. Admitted to Hammersmith Hospital under the care of Professor Grey Turner. Complained of the presence of a lump on his bladder, which had been exposed from birth, and of pain in the small of the back.

HISTORY.—His bladder had been exposed to the surface from birth, and he had no control over his water, which at all times trickled over his pubis, soaking his clothes and leading to excoriation of the inner side of the thighs. When a baby he had had an operation at Great Ormond Street for the bladder condition, but he did not know what had been done. He was not allowed to go to school, and so could read only a few words, and could not write except to sign his name. He said "My mother was frightened by a cow. That was the cause of it". He wore skirts till the age of nine when he commenced to wear trousers; these had to be worn low because they hurt him. During his young days he played about at home. The bladder and excoriated areas were washed by his mother each week-end when his father was at home to hold him down. At about 15 years of age he began to resent his mother washing him, and did it himself daily, finding that this was less painful and did not allow the area to become coated. He was then developing normal sexual desires and had noctural emissions. He commenced to work as a farm-hand at a nearby farm, going home three or four times a day to change his underpants. The bladder protrusion gradually increased in size. When 36 years of age, getting fed up with the conditions, he decided to go to hospital to see if anything could be done about it. He was fitted with a bag which acted both as a shield and a container. This was very satisfactory, and stopped the irritation of the clothes. He had no longer to keep his hands in his pockets to keep the clothes off his bladder, and sitting kept quite dry unless he sat too far back on the seat. He could carry out all normal farm work and could bear 1-cwt. loads on his shoulders, but was unable to swing them into that position from the ground. He had continued work until three years ago, when he had to give it up because he developed backache. The weather affected his back, rain and south wind heralding the pain. Two years ago he noticed that a lump, which looked like a wart, had appeared on the bladder area. He went to a hospital and was told that nothing could be done about it. The lump had gradually increased in size, it bled if he strained, and sometimes bled spontaneously. There was no pain in the lump, but this occurred if it were pressed against the underlying bladder. He had no pain in the loins or shivering attacks; the urine in the bag was quite clear, but thickened in the evening and also in hot weather.

His general health had been good, he had had no headaches, and had a good appetite and slept well.

Family History.—He stated that his father and mother "died of old age". One brother and three sisters were all alive and well. There was no history of any congenital abnormality in his antecedents.

ON EXAMINATION.—He had the typical appearance of a ruddy-faced farmworker, but with a waddling gait and a strong uriniferous odour. Examination of the lower abdomen revealed the condition as seen in the accompanying photograph (*Fig.* 23). This

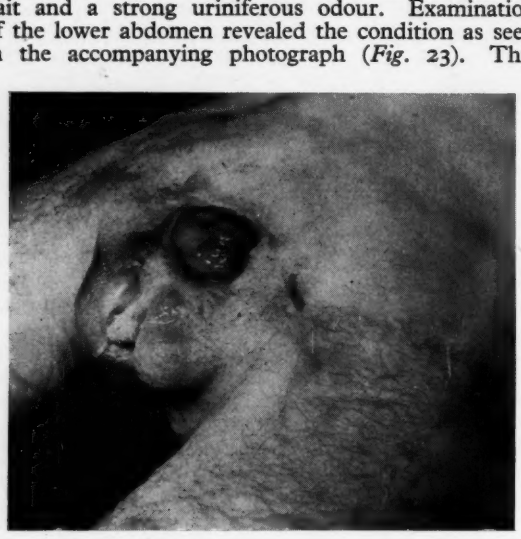

FIG. 23.—*Case* 1. Photograph of complete ectopia vesicæ with malignant ulceration present in upper left quadrant.

was a typical case of complete ectopia vesicæ. The umbilicus was absent, as was the hypogastric area of the abdominal wall, which was occupied by the red, bulging mucous membrane of the posterior wall of the bladder. The upper left-hand quadrant of the exposed mucous membrane was the site of a plaque-shaped, ulcerated area, measuring 1 in. by 1½ in. The lower part of the bladder was bridged by a flap of skin under which was a pool of urine. The whole of the mucous membrane was very sensitive, and at the upper rim of the posterior border of the pool the ureteric orifices could be seen to be intermittently ejecting urine. Below the bridge of skin was to be seen an epispadic penis lying between the two halves of a bifid scrotum containing normal-sized testicles. Two lateral prominences, some 4 in. apart, were the separated pubic bones, joined by a strong ligament lying under the skin bridge. This skin bridge suggested that the operation performed in childhood was the one devised by Woods, which attempted to provide a covering for the exposed bladder with flaps of skin. The perineum was broadened (4–6 in.) and the whole pelvis widened. The great trochanters of both femora were felt to be well above Nélaton's line. On rectal examination, no prostate or vesicles could be felt; the thick interpubic ligament could be palpated and the bladder wall made to bulge by the examining finger. There were no enlarged glands in either groin. Examination of the other systems revealed no abnormality and there was no evidence of secondary deposits. A diagnosis was made of ectopia vesicæ complicated by malignant ulceration.

Investigations :—

Blood-urea, 42 mg. per 100 c.c.

Intravenous urography showed both kidneys to be excreting. The renal outlines were of average size, but both were low in position. The pelves were of average size and there was no contoural deformity of the calices.

Radiography of the pelvis showed congenital deficiency of the anterior wall of the pelvis, the pubes being widely separated, and the ilium splayed out. In the right hip there was chronic arthritic change. There was a slight lipping of the dorsilumbar spine.

It was decided to remove the bladder before the transplantation of the ureters, in order not to waste any further time in the treatment of a malignant condition. Further, the way in which he stood the operation would be an index to his renal capacity; should he not do well after excision of the bladder, then the transplantation would not be attempted.

OPERATION.—Cystectomy of the ectopic bladder was carried out by Professor Grey Turner. The skin-flap was divided down the centre and the incision was extended round the circumference of the exposed mucosa. The mucous membrane, including the growth, was dissected up from the peritoneum in the upper part and from a dense fibrous tissue towards the pubic area. As frequently happens, the peritoneum was opened and this presented an opportunity to palpate the abdominal glands. Free bleeding occurred as the corpora cavernosa of the penis were cut across, but this was controlled by hæmostatic sutures. Further hæmorrhage was encountered in the region of the ureters, the vessels being clamped and tied. The ureters were divided as they entered the under surface of the bladder; they were noted to be of normal size, about the thickness of a goose quill, and were secured against retraction by transfixing them to the side of the wound. The previously divided skin-flap was reconstituted and the exposed bare area packed with dry gauze, which in its turn was covered with flavine-soaked gauze.

PROGRESS.—The patient made a steady and uneventful convalescence. The granulating surface tended to become covered with phosphatic deposits, which was overcome by increasing the acidity of the urine with mist. acid soda phos. and by washing the

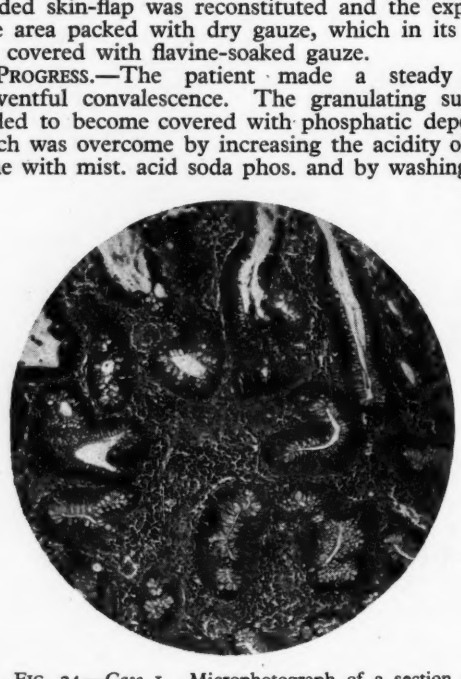

FIG. 24.—*Case* 1. Microphotograph of a section of the adenocarcinoma. Consists of tall columnar-celled epithelium of a mucin-secreting type. The cells are largely goblet cells and are distended with mucoid secretion.

area with dilute acetic acid. Following this the phosphatic crusts could be gently peeled off and the wound gradually healed in. Within six weeks his general condition was very satisfactory and he could again wear his apparatus and was fit to return home.

He was readmitted eight months later (July, 1941). His general condition was very satisfactory. The ureters were transplanted into the sigmoid colon. He made a good recovery.

PATHOLOGICAL EXAMINATION.—A portion of the tumour (*Fig.* 24): This is a rather polypoid adenocarcinoma. The epithelium everywhere consists of tall columnar-celled epithelium of a mucin-secreting type which very closely simulates, and is in fact indistinguishable from, a rectal columnar-celled carcinoma. The cells are largely goblet cells and are distended with mucoid secretion.

TYPES OF TUMOURS FOUND IN ECTOPIC BLADDERS

The histological examination prompted the investigation into the type of tumours likely to be found in the ectopic bladder.

In Normal Bladders.—Tumours of the adenomatous type are rare in the normal bladder. Watson found 2 simple adenomata in a series consisting of 653 bladder tumours, and Geraghty 1 in 145. Arising from accessory prostatic glands which are to be found behind the internal urethral orifice and in the region of the inter-ureteric bar (Jores and Thorel), tumours have been reported attaining the size of an apple. These glands exist at all ages and are independent of prostatic affection. They show an acinar structure lined by cubical cells and in places exhibit papillary formation and may contain prostatic concretions. Clado has also shown vesical adenomata not resembling prostatic tissue. Verhoogen and Steinhaus consider that there are other glands of bladder origin shown histologically to be lined by high cylindrical cells. These glands may undergo cystic change, and in their vicinity may be seen unaltered glands or glands in an early stage of cystic formation. Albarran considers that they are of allantoic origin, but they are rare. Lendorf states that there are no epithelial downgrowths in the embryonic bladder, that in childhood solid buds develop, and later in life definite simple glands.

Adenocarcinomas form 2 per cent of bladder tumours. Scholl found 5 in a series of 323. Saphir and Kurland, in reviewing the literature, found 22 cases arising in the bladder itself and added 3 further cases, one of which arose from the apex, which type will be discussed below. They summarized the various opinions as to the origin of the tumours as arising from: (1) Glandular structures supposedly normally present; (2) Remnants of lining cells of the cloaca and of the sinus urogenitalis; (3) Misplaced intestinal mucosal cells; (4) Remnants of the urachus; (5) Cystic structures occurring in instances of chronic cystic cystitis.

It is at the apex of the bladder that we find tumours most closely resembling those found in the ectopic bladder. De Korte describes a tumour arising from the apex which grossly and histologically resembles an adenoma of the intestine, and Kaltenbach a tumour arising from the mucosa near the apex which microscopically resembles colloidal carcinoma of the rectum. Nicholson, discussing heteromorphoses, describes a tumour in the unobliterated lower end of the urachus consisting of tubules and cystic spaces lined by irregular cubical and columnar epithelium. He considers this to be prostatic tissue, and concludes that all the normal glands of the cloaca, as well as those formed in pathological conditions, are of one type—namely, prostatic glands. Any part of the cloaca failing to undergo its normal disappearance is thus able to become differentiated into identical glands. This potentiality is shared by the mesodermal Wolffian duct that forms the trigone of the bladder. Not only are these cells able to persist, but they retain the same prospective potentialities as those of the permanent parts of the cloaca. Campbell Begg has made an extensive study of these apical tumours, and describes 21 adenocarcinomata in this region, all closely resembling carcinoma of the rectum. He states that they may originate in that part of the urachus contained in the apex of the bladder. Saphir and Kurland remark that whereas urachal adenocarcinomas show only glandular structures, adenocarcinomas of the bladder itself show not only adenocarcinomatous but also transitional carcinomatous-cell structure. It is interesting to note that the urachus is the drawn-out upper part of the primitive bladder and corresponds to the upper part of the ectopic bladder which has been seen to be the site of similar growths. As a further point of comparison, in a very few cases the apex of the bladder and the ectopic bladder have been the site of squamous epitheliomata. Discussing the urachal tumours, Begg considers that "the retention by urachal cells of the potentialities of primitive cloacal cells, and their ability to bring them at any period of life to untimely fruition, are the keys to the understanding of the various types of tumour of the urachus. Complete, but ill-regulated, development towards glandular or transitional epithelium respectively may be observed, may overstep all bounds, and give rise to colloidal carcinoma on the one hand or squamous-celled carcinoma on the other. Both these types were seen in a single tumour in the sixth case of Rankin and Parker. As a rule, however, the full impulse given by the entodermal origin of the cells predominates, and the formations commonly seen pass through the stages of simple adenoma and adenocarcinoma to reach finally the fully formed colloid cancer of the rectal type".

Many authors consider that the fundus of the bladder possesses no glands, any that are present being of prostatic origin (Virchow), urethral (Aschoff), cloacogenic (Endelman and François), or the result of inflammation. The consensus of modern opinion is that there are no glands present normally in the mucosa of the corpus of the urinary bladder. This is endorsed by Nollendorf. With long-standing infection, metaplasia of the mucosa follows, cystic formation

occurring both in the mucosa and submucosa. This is the condition described as cystitis cystica. Morton (1925) suggests that this condition is closely related to cystitis glandularis, which is produced by the rupture of these cysts due to continued chronic infection, and the development of true mucous glands with goblet cells and the typical structure of tubular glands such as are normally present in the intestine. Stoerk, Zuckerkandl, and Albarran have shown the metaplastic potentialities of vesical epithelium proceeding from cystitis cystica to fully formed colloidal adenocarcinoma. In an excellent article on this subject in 1935, Patch and Rhea point out that cystitis cystica occurs much more frequently than is generally recognized. In fact, Morse, at the Mayo Clinic, in the cystoscopic examination of 190 cases of bilateral pyelonephritis, noted cystitis cystica in 33 (17·4 per cent). It may develop in the course of any long-standing inflammation not so intense as to destroy the epithelium. In particular, the presence of stone or the inflammatory exudates from infected kidneys have been met most frequently in association with the cystic and glandular formations. Eight of the twelve cases studied by Patch and Rhea had aseptic chronic urinary retention, secondary to adenomatous enlargement of the prostate. Clinically the cysts appear as small, round, transparent vesicles up to 5 mm. in diameter, and not unlike the nodules seen in bilharziasis. Morgagni (1822), Rokitansky (1869), and Litten (1876) noted and described these cystic formations. But the present conception of their origin is due to the work of von Limbeck (1887) and von Brunn (1893). According to them, buds from the deeper layers of the thickened mucosa project into the submucosa and are pinched off. These are commonly known as 'Brunn's nests'. Von Brunn considered that the cystic formation was begun by central degeneration in these nests. Patch and Rhea, however, agreed with Stoerck and Zuckerkandl that the central cysts are the result of secretory action. They studied serial sections stained with muci-carmine from the cases of cystitis cystica and glandularis, and came to the conclusion that " there can be seen in the earliest stages of the development of the cyst, a centrally placed, small, red droplet. From this beginning, the changes in the epithelial nests develop in one of two ways. The cysts may increase in size at the expense of the epithelial cells which comprise them, until cysts are produced the linings of which are composed of flattened epithelial cells surrounding a colloid-like substance—the fully developed cystitis cystica. On the other hand, the epithelial cells surrounding the early cysts, may become tall, mucus-secreting cells with basally-placed nuclei." Whether the cystic formation originates from degenerative or secretory processes at the centre, the interesting point, as Saphir and Kurland point out, is that the adjacent cells change from transitional to columnar in type, which fact explains the presence of glandular tissues and the occurrence of adenocarcinoma. The change itself is difficult to explain. H. Pincsohn, quoted by Saphir and Kurland, considers that the columnar epithelium develops by means of " indirect metaplasia ". The dominant cell type of a tissue may become inferior to the recessive cell type which in turn becomes dominant. Due to irritation the transitional cell of the bladder reverts to the recessive type, and the inert recessive type, the cylindrical cell, becomes dominant, hence columnar epithelium is originated.

In Ectopic Bladders.—The structure of the wall of the ectopic bladder presents quite a different appearance from the normal bladder. Vrolik (1922) first remarked on the curious mucous covering of the ectopic bladder, and Lichthein (1873) mentioned the complete glandular covering observed by him. Superficially there is thickening and keratinization of the epithelium, resembling epidermis, whilst in the deeper layers is glandular tissue formed by high cylindrical epithelium, with mucus-containing goblet cells as shown in *Fig.* 24. This glandular formation, which has been noted in all the cases which have been subjected to microscopy, would appear to be usual to the ectopic bladder, whereas it is exceptional in the normal bladder. Thus the high incidence of adenocarcinoma of the intestinal type in the ectopic bladder is accounted for. As has been noted above in 25 cases subjected to histological examination, 23 were adenocarcinomata and 2 were malignant epitheliomata. The origin of the squamous epitheliomata or of the possible origin of the double tumour, is to be seen in the superficial stratified and the deep glandular layers.

Several hypotheses have been advanced which might account for the appearance of the adenomatous tissue. Enderlein examined the bladders in 2 newborn ectopics, and found neither glands nor cylindrical epithelium, but normal transitional epithelium. In 18 cases, examined by Shattock, Enderlein, and Scholl, ranging from a few months to 77 years of age, glandular tissue was present. Enderlein considers that the appearance of the glandular tissue is due to the many traumata such as exposure to air and drying, and irritation by clothes and infection, to which the mucosa of the ectopic bladder is necessarily exposed. Ehrich expressed the belief that the glands develop from a displaced or undeveloped spur of cloacal entoderm, whereby embryonic epithelium of the dorsal or rectal wall of the cloaca is included in the ventral vesical wall. In addition, he considered that there is a lack of stimulus to assume normal transitional epithelium. Reichel considers it to be a failure of the cloacal epithelium to differentiate normally as vesical epithelium, and thus contains glands like those in rectal mucosa. Righetti studied the rectums of exstrophics and found areas of vesical epithelium and other areas of undifferentiated

cells, evolving at certain points to vesical and at others to intestinal cells. Remedi declared that certain glands from substrata foreign to the bladder or rectum become abnormal at an early period of life for an unknown reason, and persist throughout life. Hovelacque and Lacene prefer to see it simply as the true ectopy of undifferentiated cells from the primitive intestine, transformed before the appearance of the malformation on to the bladder mucosa. Nicholson considers this heterotopic columnar epithelium to be the best example of an extremely rare metamorphosis from a transitional to columnar epithelium, and quoting Schridde states that the higher differentiation of the columnar epithelium over the transitional epithelium renders such a metaplasia less liable to occur. Judd and Thompson, from embryological considerations, think that the glandular tissue is a result of cell inclusion. The intestinal fistulæ complicating exstrophy as reported by Keith, Hache, and von Geldern, demonstrate the close relationship between the intestine and the bladder developmentally. Ewing states that the study of many adenocarcinomata indicates that the pure adenomatous structure may represent an intermediate or transitory stage in the evolution of the tumour, which is rapidly traversed and soon passes into a more typical carcinomatous stage. Scholl considers it is a combination of metaplasia and hyperplasia of normal glands often seen in the mucosa of the bladder. He examined 9 exstrophied bladders at the Mayo Clinic in an effort to establish connexion between the constantly present hyperplastic mucosa and the glandular malignancy which so often develops. The mucosa contained areas gradually shading off from fairly normal to definitely malignant tissue. The youngest, aged 4 years, showed a moderate amount of normal epithelium still to be present and the gland formation only slightly advanced. Such formation seems to vary with the age of the patient, but a child 6 years old was an exception in showing much glandular tissue.

Table I.—Brief Particulars of 26 Reported Cases of Ectopia Vesicæ complicated by Carcinoma.

Case	Year	Author	Sex	Age	Lesion	Treatment	Comment
1	1895	Bergenhem (Lacene and Hovelacque)	M.	35	Adenocarcinoma	Transplantation of ureters, extirpation	Working 6 months after operation
2	1901	Ehrich	F.	44	Adenocarcinoma	Resection, transplanted ureters and bladder mucosa	Died 8th day from ascending infection
3	1904	Enderlen	F.	38	Adenocarcinoma	Radiotherapy	Not benefited
4	1905	Lampe	M.	—	Squamous-cell carcinoma	Sonnenberg operation	Recovered not treated
5	1906	von Elselberg	F.	54	Adenocarcinoma	Extirpation	Living 15 months after operation
6	1909	Sargent	M.	39	Clinically carcinoma	None	Not traced
7	1910	Wagner	M.	49	Adenocarcinoma	Biopsy only	Not traced
8	1910	Hager	M.	66	Adenocarcinoma	Symptomatic	Died from cachexia
9	1910	Schloffer	M.	46	Carcinoma	Curettage	Result not mentioned
10	1912	Lacene and Hovelacque	M.	48	Adenocarcinoma	Symptomatic	Died from cachexia
11	1912	Lacene and Hovelacque	F.	57	Squamous-cell carcinoma	Previous nephrectomy	Died from cachexia
12	1914	Hunner	F.	26	Adenocarcinoma	Not mentioned	Result not mentioned
13	1918	Geraghty	—	48	Adenocarcinoma	Not mentioned	Result not mentioned
14	1921	Lower	M.	50	Clinically carcinoma	Transplantation of ureters; excision	Died: abdominal metastases
15	1922	Scholl	F.	23	Adenocarcinoma	Transplantation of ureters; excision	Died 2 years after operation from metastases
16	1922	Scholl	M.	48	Adenocarcinoma	Excision; radium	Reported well 6½ years later
17	1922	Dupont	M.	38	Adenocarcinoma	Exclusion of rectum	Died 2 days after operation
18	1924	Murphy	M.	49	Adenocarcinoma	Radium emanation	Examined after 14 months
19	1925	McCarthy and Klemperer	M.	47	Adenocarcinoma	Diathermy; ureterostomy; nephrectomy; cystectomy	Convalescent at time
20	1928	Judd and Thompson	F.	38	Adenocarcinoma	Cautery excision of bladder	Well 3 years after operation
21	1929	Hammer	M.	60	Adenocarcinoma	No treatment; uræmic death	No metastases found
22	1929	Hammer	F.	55	Adenocarcinoma	Uræmic death	No metastases found
23	1930	Scheuer	M.	39	Adenocarcinoma	Removed electro-cautery; X-ray therapy; healed after 5 months	Note: this patient had had plastic operation to re-form bladder at 3 years of age
24	1935	Gayet	F.	64	Adenocarcinoma	Refused treatment	Lost contact after 1 month
25	1931	McCown	M.	62	Adenocarcinoma	Bilateral lumbar ureterostomy; diathermic partial removal	Died: operative shock; autopsy showed no metastases
26	1934	Montpelier, Goinard, Karsente, and Mele	M.	40	Adenocarcinoma	No treatment	Died few days after admission

RÉSUMÉ OF OTHER REPORTED CASES

(See Table I)

The case reported by Bergenhem (1895) was a man of 35 years. A sanious ulceration had developed on the mucosa of an ectopic bladder. The ureteric orifices were normal. The bladder was excised, and the ureters were implanted into the rectum (Peters' operation). Biopsy showed the cylindrical malignant adenoma of the mucosa. The patient recovered, and when seen six months later was at work and could control the urine in the rectum. This would appear to be the ideal method of dealing with these cases, but it was not until 1921 that it was again successfully performed by Lower. In 1905 Lampe successfully excised the bladder and transplanted the ureters into the epispadic urethra (Sonnenberg operation). The patient was not followed up. Percy Sargent's case was of a man of 39 years who had developed an epithelioma round one of the sinuses resulting from a plastic operation performed 35 years previously. Because of enlarged hard glands in both groins, the case was considered inoperable. No biopsy was done. Sargent considered that this possibility of malignancy afforded addition to the many reasons which exist for abandoning the unsatisfactory plastic method of dealing with extroversion of the bladder. In 8 cases symptomatic treatment only was given, as the condition was considered to be too advanced, not from the presence of metastases, but from the general condition of the patient and the extent of the local area involved. In 2 of these cases, reported by Lacene, post-mortem examination revealed no metastases. Von Elsberg excised the bladder in his case, and the patient was living fifteen months later. In 1922, Scholl dealt successfully with 2 cases. In the first he excised the bladder and transplanted the ureters into the rectosigmoid colon; in the second case he excised the bladder and applied radium. The first case lived two and a half years, dying from metastases, and the second was well six and a half years later. Murphy employed radium emanation, and the patient was alive fourteen months later. McCarthy used diathermy and then excised the bladder. His patient was still convalescing at the time of the report. In 1928 Judd and Thompson reported 2 cases. In the first, cautery excision of the bladder was carried out, and the patient was well three years later; the second case was inoperable. Scheuer used the electro-cautery, followed by X-ray therapy. The bladder area healed after five months. Hammer described 2 cases which were considered too advanced for operative measures. Both died from uræmia. No metastases were found at post-mortem examination. Dupont's case died two days after exclusion of the rectum, which he had performed as a first stage in transplantation of the ureters. Post-mortem examination revealed no metastases. In April, 1940, McCown reported a very advanced case on which he operated in 1931, and which died shortly after operation, due to shock. Once again post-mortem examination revealed no metastases. This low malignancy of these growths, as shown by the absence of metastases at post-mortem examination, must be noted. In 3 cases the inguinal glands showed definite clinical involvement, but microscopical confirmation was lacking. It should be an encouragement to undertake radical treatment even in the most advanced cases. The following case which I am allowed to quote by the kindness of Mr. Henry H. Wade, of Edinburgh, demonstrates most strikingly that these tumours may attain very large proportions and yet remain local.

Case 2.—Mr. C., male, aged 53 years. This man had had complete ectopia vesicæ from birth. The typical congenital deformity of the pelvis is shown

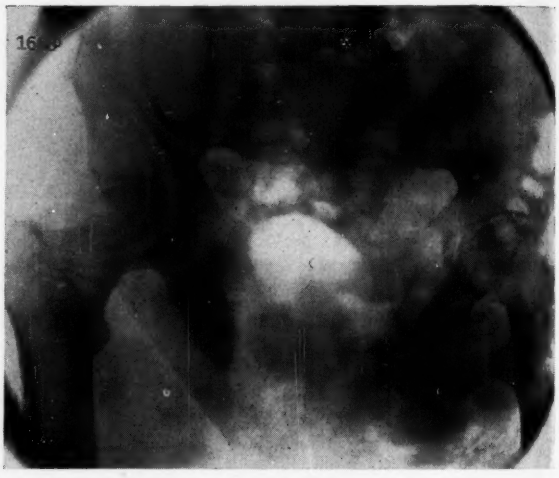

FIG. 25.—*Case* 2. Radiograph of the pelvis, showing the characteristic deformities of this congenital malformation.

in *Fig.* 25. For two years he had noticed a tumour on the extruded bladder wall steadily increasing in size. At the time of presenting himself for surgical advice an extensive fungating growth (*Fig.* 26) occupied the larger part of his abdominal wall and was so foul smelling that it was more in the interests of the relatives than the patient that he was seen.

EXAMINATION.—Radiograph of the pelvis showed the characteristic deformities of this congenital malformation (*Fig.* 25). Intravenous urography showed a very extreme degree of dilatation of the ureters and renal pelves.

AT OPERATION.—The large cancerous growth was widely excised, a procedure necessitating the removal of a large portion of the external wall of the abdomen. The ureters were divided and left undisturbed and allowed to form natural fistulæ where they lay. These fistulæ ultimately came, after the bladder had been removed, to discharge on to the abdominal surface. The repair of the defect of the abdominal wall was successfully accomplished. The urine is voided into a depression in the lower part of the abdomen, into which both ureters open. The ureters in this case

were so distended that transplantation into the bowel was impossible.

In a recent personal communication, Mr. Henry H. Wade informs me that this patient is alive and well

FIG. 26.—*Case* 2. Drawing showing ectopia vesicæ complicated by an extensive fungating adenocarcinoma.

and comfortable; he has resumed work and now (four years after operation) has no recurrence of malignancy.

FIG. 27.—*Case* 2. Low-power microphotograph of section of adenocarcinoma, showing tall columnar mucin-secreting epithelium.

PATHOLOGICAL EXAMINATION.—A portion of the tumour (*Fig.* 27): This is a polypoid adenocarcinoma, lined by tall columnar-celled epithelium.

DISCUSSION

In order to verify the statements and, if possible, to offer some explanation as to the origin of the adenomatous tissue, a short series of ectopic bladders of varying ages has been collected and histological examination carried out. The youngest, aged 4 months (*Fig.* 28) shows a transitional-cell lining, hyperplastic and keratinized in places. Transitional-cell epithelial nests are to be seen deep to the hyperplastic lining, and one has undergone cystic change. *Fig.* 29, from the same sections, shows a larger

FIG. 28.—Low-power microphotograph from section of bladder of 4-months-old ectopia vesicæ. The mucosa is hypertrophied and its surface keratinized. In the submucosa are several transitional-cell nests, one showing cystic formation.

cyst in which the lining, although transitional, is somewhat modified. The 6-years'-old specimen (*Fig.* 30) has a lining which is in places

FIG. 29.—Higher-power view of a cell nest from same section as in *Fig.* 28. The transitional-cell lining is somewhat modified in places, approaching a columnar type.

keratinized and squamous in character. There are numerous cystic spaces, some lined by transitional epithelium, others by a single layer of flattened epithelial cells. One area (*Fig.* 31)

shows glandular tissue lined by tall columnar-celled mucin-secreting epithelium. Two specimens (*Figs.* 32, 33), aged 45 years and 48 years

FIG. 30.—Low-power microphotograph of section of bladder of 6-years-old ectopia vesicæ. 'Cystitis cystica'—numerous cystic spaces, some lined by transitional epithelium, others by a single layer of flattened epithelial cells.

respectively, showed a similar lining, but the submucous layers contained a much larger proportion of glandular tissue. The older specimen (*Fig.* 33) was from the healthy part of the bladder of *Case* 1. It is possible to find areas in which some cells of the mucous type occur amongst squamous cells in these glands, suggesting that

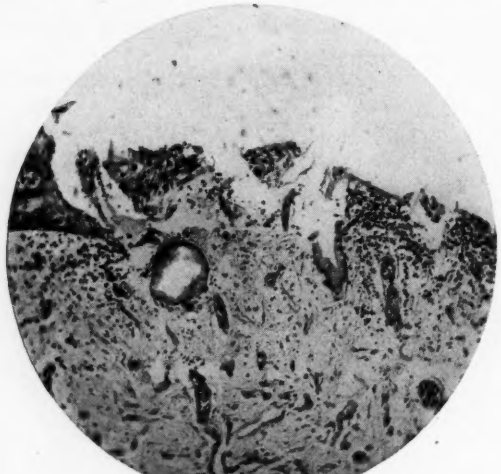

FIG. 31.—From same section as *Fig.* 30. 'Cystitis glandularis'—in the submucosa are two areas of glandular tissue lined by tall columnar-celled mucin-secreting epithelium.

one type of epithelium is transformed into the other as a result of local environmental conditions. Other glands of the mucous type are not fully differentiated in this direction, but show evidence of transformation and intermediate stages between mucous and squamous epithelium. Thus from the earliest months of life the development of cystic and glandular areas is seen commencing, as transitional-cell epithelial buds, and passing

FIG. 32.—High-power microphotograph of section of bladder of 45-years-old ectopia vesicæ. The mucosa is keratinized and squamous in character. In the submucosa is an abundance of glandular tissue showing mucous goblet cells.

through the various stages until fully formed columnar-celled mucin-secreting epithelial-lined glands—the precursors of adenocarcinoma—are reached.

Embryologically the bladder is derived from two sources: the body, lying above the interureteric bar, originates from the entodermal cloaca; the trigone is developed from the mesodermal Wolffian ducts. Were the adenomatous

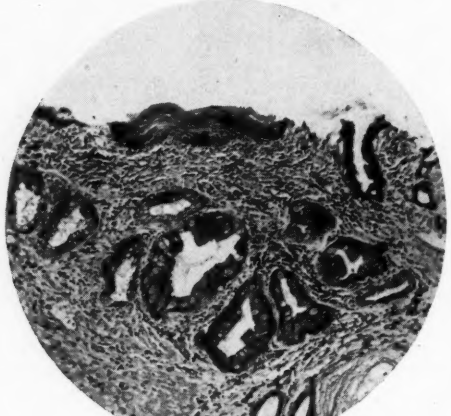

FIG. 33.—*Case* 1. High-power microphotograph from section of healthy part of bladder wall. Again the squamous-celled lining and abundant glandular tissue in the deeper layers are to be noted.

tissue of embryological origin of cloacal cell-nests one might expect to find it limited to the body of the bladder. Histological examination, however, shows that it is as profuse in the trigone as in the body. Similar pathological changes are occasionally seen in the ureter and pelvis of the

kidney, known clinically as ureteritis cystica and pyelitis cystica respectively. There is no apparent reason to differentiate between the transitional epithelium of the ectopic bladder and the normal bladder and ureter. Therefore, it is considered that the adenomatous tissue of the submucous layers of the ectopic bladder is derived from a metaplasia of the normal transitional epithelium lining brought about by the irritation to which it is exposed. Saphir and Kurland have remarked on the transitional-cell carcinoma present in adenocarcinoma of the normal bladder. Although transitional-cell tissue is to be seen in the earlier stages, no transitional-cell carcinoma is to be seen in the two cases of adenocarcinoma of the ectopic bladder under discussion. *Fig.* 34 is a

FIG. 34.—Microphotograph of section of adenocarcinoma of anatomically normal adult bladder. The growth is identical with that seen in *Figs.* 24 and 27.

microphotograph of a tumour removed from the fundus of a bladder and is an adenocarcinoma identical with the adenocarcinoma seen in the two ectopic bladders. Again no transitional-cell carcinoma is to be seen. As regards treatment, the glandular tissue present in the mucous membrane of the ectopic bladder is undoubtedly of a precancerous nature, indicating that in all cases the bladder must be removed. Where malignancy has already developed no time should be lost in undertaking surgical measures. The ureters may be left draining temporarily on to the suprapubic region. Continence, however, may be achieved by transplantation of the ureters into the colon. The modern simple technique and low mortality, in all except advanced malignant cases, as shown by the series of Grey Turner, Wade, Ward, Morson, and Graham in this country, and several large series in the U.S.A., fully justifiy the undertaking of this procedure.

SUMMARY

1. Two cases of ectopia vesicæ complicated by adenocarcinoma are presented.

2. It is an uncommon condition, but in 23 out of 25 cases subjected to histological examination, the growth has been an adenocarcinoma closely resembling adenocarcinoma of the large intestine.

3. The literature of adenocarcinoma of the bladder is reviewed, including the bladder proper, the apical growths probably of urachal origin, and the ectopic bladder. The various hypotheses as to the origin of the adenomatous tissue are noted.

4. The development of the tumour is traced from the buds of transitional-cell epithelium from the deeper layers of the hyperplastic lining epithelium, which become cystic, giving rise to cystitis cystica. The lining cells of the cysts then change into cylindrical cells, thereby causing cystitis glandularis.

5. The adenomatous tissue in the ectopic bladder is of a precancerous nature and the bladder must be removed.

6. The growth remains locally malignant until very advanced, and metastases are rare.

7. The operation of choice is excision of the bladder preceded or followed by transplantation of the ureters into the rectum.

I should like to acknowledge my indebtedness to Mr. Henry H. Wade for permission to use his case and for the loan of the beautiful coloured drawing; to Professor J. R. Learmonth for the microscopical sections and photograph of the same specimen from the Museum of the Royal College of Surgeons of Edinburgh; to Mr. F. J. F. Barrington for the specimen in *Fig.* 34; and to the Curator of Guy's Hospital Museum for the specimen in *Fig.* 28. In particular, I wish to thank Professor J. H. Dibble for his generous advice on the various pathological sections, and to Professor Grey Turner, who first suggested this investigation, for permission to utilize his case, and for his most valuable advice and interest in the preparation of this article.

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* These articles report the cases listed in *Table I.*

THE MORBID ANATOMY OF A CASE OF RECURRENT DISLOCATION OF THE SHOULDER

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THE literature of recurrent dislocation of the shoulder has of late been very largely given up to the discussion of various operations, and of the results obtained from these.

Much less attention has been given to the morbid anatomy. In this country, Blundell Bankart has been in the foreground in this province, and contends that the essential lesion is the detachment of the capsule, with or without the glenoid labrum, from the anterior lip of the glenoid cavity. He further maintains that the lesion is a direct anterior dislocation produced by direct violence to the shoulder or arm, whereby the head of the humerus is caused to dislocate anteriorly. It is thus quite different from the simple anterior dislocation of the shoulder, which occurs with the arm in abduction.

In this connexion it is interesting to note that Peterson has recently described a first dislocation of the shoulder in an epileptic, noting that this occurred without any abduction of the arm taking place.

Codman, in his book on the shoulder, gives a most comprehensive list of causes of recurrent dislocation of the shoulder, without selecting any morbid anatomical feature as the usual explanation of the recurrence.

Tavernier's article in the *Revue d'Orthopédie* in 1929 includes a very complete review of the morbid anatomy. He quotes freely from Alibert (*Thèse de Paris*, 1920) and concludes that the morbid anatomy consists of:—

1. A capsular detachment from the anterior rim of the glenoid cavity, with or without the glenoid labrum, and even occasionally with a rim of bone.

2. In addition, some abnormality of the head of the humerus: (*a*) The posterior notch; (*b*) The hatchet deformity of the head.

To quote a translation from Tavernier, "We have insisted on the part played by malformations of the head (notch and hatchet deformity), which are the better known because they are the more easily observed, but there would be nothing astounding in an insufficiency of the glenoid cavity, more difficult to demonstrate, but which has been noted several times (2 cases by Turner Thomas, 1 case by Goldmann), one day taking its place beside the deformities of the head in explaining the recurrence of the dislocation."

Following this introductory reference to the literature, we will now proceed to describe the case.

CASE REPORT

HISTORY.—A woman, of 36 years of age and an epileptic, first suffered a dislocation of her right shoulder during a fit some nine years ago. Her shoulder was out for some hours, as was the case on three or four subsequent occasions. An anæsthetic was not necessary for the reduction of the shoulder on any occasion, although the doctor was called in frequently. During the last three years the shoulder had been out about twenty-five times. Dislocations, while first occurring only with fits, had of late occurred frequently with trivial exertion, as, for instance, when putting on her coat. There had also been some pain in the shoulder without dislocation actually occurring. In view of the frequency of the dislocations, an operation was advised.

OPERATION.—Under the anæsthetic it was found that the head of the humerus could easily be pushed forward, this resulting in a dislocation without the arm being in any way abducted from the side. A Nicola operation was performed and no lesion was detected through the limited exposure used for this operation. As the operation drew to a close the anæsthetist said that the patient's heart had stopped. Cardiac massage and other means failed to restart the heart.

POST-MORTEM EXAMINATION.—A more extensive examination of the front of the shoulder-joint revealed the lesion of the capsule and glenoid labrum illustrated and described later.

It was only after opening the capsule posteriorly that the large notch was demonstrated; this is also illustrated and described later. No loose bodies were found in the joint.

DISCUSSION

THE ANTERIOR CAPSULE

Anatomy.—The glenoid labrum is a fibro-cartilaginous rim attached to the margin of the glenoid cavity. It is triangular in section, with a free edge, and is attached by its base, thereby deepening the glenoid cavity.

The anterior capsule shows two strong ligaments, the superior and inferior gleno-humeral ligaments, which embrace the orifice of the subscapular bursa. Both these ligaments are attached to the upper part of the anterior margin of the glenoid cavity, close to the origin of the long head of the biceps. The superior ligament passes along the median border of the tendon of this muscle, to be attached to a depression upon the lesser tuberosity, while the inferior ligament passes more obliquely downwards to gain attachment to the lower portion of the anatomical neck of the humerus. These points are illustrated in *Fig.* 35.

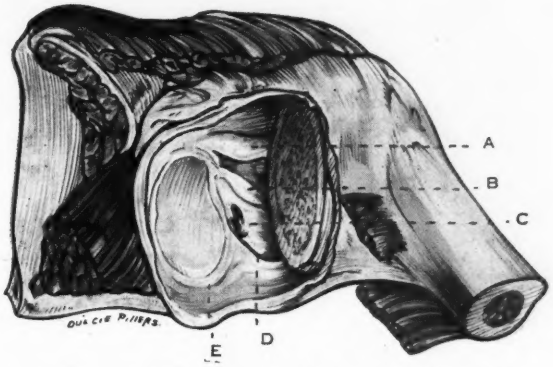


FIG. 35.—Showing the anterior capsule of the shoulder-joint from within. The head of the humerus has been removed to improve the exposure. A, Tendon of long head of biceps; B, Superior gleno-humeral ligament; C, Orifice of subscapular bursa; D, Inferior gleno-humeral ligament; E, Glenoid labrum.

In the twenty-one shoulders dissected to demonstrate this region, one was impressed with the strength and size of these ligaments in post-mortem specimens. Their description as weak bands is perhaps an illusion produced in the fixed anatomical specimens of the dissecting room.

The dissections also demonstrated that the anterior capsule is always attached to the lip

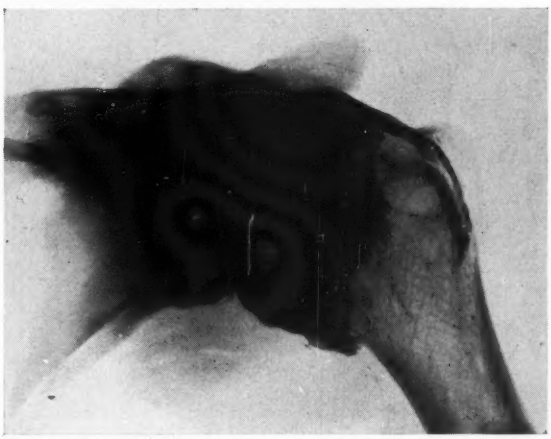


FIG. 36.—Radiograph of a normal shoulder-joint injected with lipiodol. It shows that the capsule is attached to the anterior lip of the glenoid cavity and not to the neck of the scapula. Above, the subscapularis bursa overlaps the neck of the scapula.

of the glenoid cavity and is never attached to the neck of the scapula. A joint injected with lipiodol demonstrates this point (*Fig.* 36).

A view from above the joint injected with lipiodol illustrates clearly that the opening of

the subscapular bursa is well away from the rim of the glenoid cavity and cannot therefore be confused with the capsular detachment which we are about to describe (*Fig.* 37).

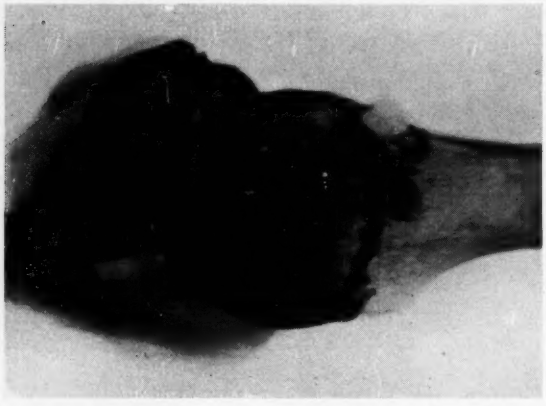


FIG. 37.—A view of a shoulder-joint, injected with lipiodol, from above. It shows the subscapular bursa with its orifice situated well away from the anterior lip of the glenoid cavity.

Capsular Lesion.—The lesion found in this case is illustrated in *Fig.* 38, and is best described by quoting Bankart when reporting on his twenty-seven consecutive cases: "In every case the anterior margin of the glenoid cavity will be found to be smooth, rounded, and free from any attachments, and a blunt instrument can be passed freely inwards over the bare bone on the front of the neck of the scapula." The depth of the pocket so formed was about 1·5 cm., save in its lower portion where it was much more shallow. The lesion had detached the gleno-humeral ligaments from the glenoid rim as it extended up to the origin of the biceps tendon and downwards as far as the inferior extremity of the glenoid cavity. The detachment of these ligaments had led to a great reduction in the anterior support to the head of the humerus, and the added space provided by this pocket would allow the humerus to come right forward.

This capsular detachment appears to have quite considerable recognition by orthopædic surgeons in many countries, and the prevalence of extracapsular operations and operations giving a very limited view of the interior of the joint, probably accounts for this recognition not being more universal.

In connexion with the detachment of the superior and inferior gleno-humeral ligaments, some operations would appear to function through the tendon of the biceps being used to reconstitute the superior gleno-humeral ligament. This is probably the case in Nicola's operation, and more particularly in Parker's modification of this operation, in which the tendon of the biceps is slid into a cleft made to extend from the bicipital groove deep to the lesser tuberosity. With a somewhat similar aim in view, Gallie creates a new inferior gleno-humeral ligament with fascia.

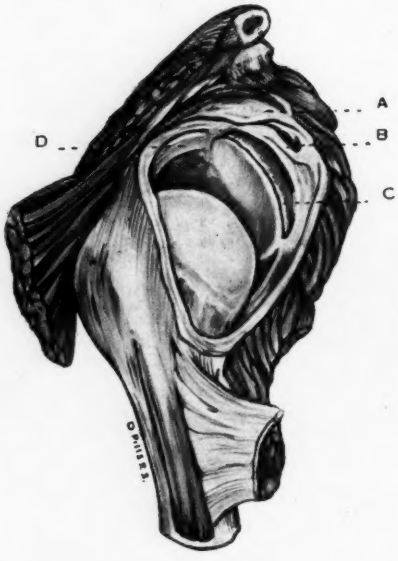


FIG. 38.—Showing detachment of the anterior capsule and glenoid labrum from the lip of the glenoid. Smooth, bare bone comprises the posterior wall of the cleft so formed. A, Superior gleno-humeral ligament; B, Inferior gleno-humeral ligament; C, Smooth bare bone and detached capsule; D, Tendon of biceps.

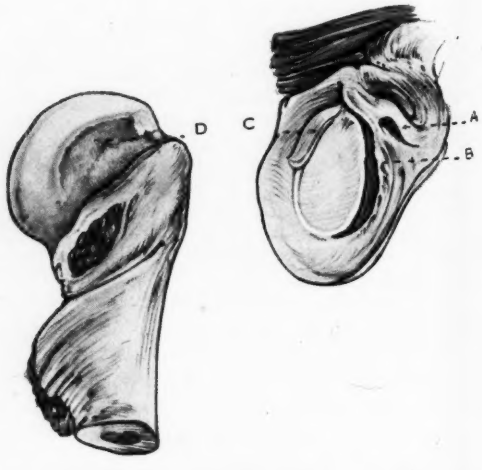


FIG. 39.—Showing the notch on the posterior surface of the head and neck of the humerus. The surface is smooth, bare bone, and is perfectly adapted to the surface of the anterior lip of the glenoid. A, Superior gleno-humeral ligament; B, Detached capsule and glenoid labrum: C, Tendon of biceps; D, Notch.

HEAD OF THE HUMERUS

The Posterior Notch.—Opening the capsule posteriorly revealed a large notch in the posterior surface of the head in this case (*Fig.* 39). The dimensions were 3 cm. in length and 1·5 cm.

across, the long axis being downwards and inwards. The depth of the notch or groove was nowhere greater than 5 mm., and its surface was smooth bare bone, denuded of cartilage, save for

FIG. 40.—Radiograph of specimen of shoulder subject to recurrent dislocation, taken from above in position of mid-rotation. Note the pronounced notch in the posterior surface of the head of the humerus, and the flattened anterior lip of the glenoid cavity compared with the overhanging posterior lip.

the upper 3 mm., where a thin layer of cartilage covered its surface.

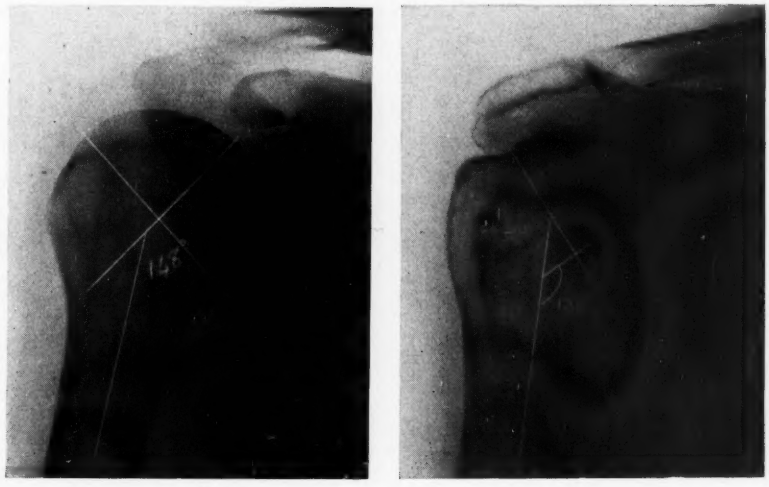
The notch in this case somewhat resembled a bicipital groove, save that its axis was not parallel with the shaft of the humerus. The notch appeared to be perfectly adapted to the anterior margin of the glenoid, and it seemed clear that these two surfaces were opposed when the head of the humerus was dislocated. In this connexion the partial disappearance of the anterior lip of the glenoid, as shown in the radiograph taken from above (*Fig.* 40), must be noted.

The older surgeons as quoted by Tavernier, described the notch in the following terms: "It appeared as if one had removed a portion of bone in the shape of a quarter of an orange." Tavernier states that the dimensions appear to vary between Perthes' description, "analogous to the bicipital groove", and a size so great that a resection of the joint is the only operation that can be contemplated (Picqué).

Tavernier states that the relationship between the prevalent type of operation and the recognition of the lesions in recurrent dislocation of the shoulder is further brought out by the fact that the notch was widely recognized when resection of the joint was the only operation employed for the condition (Cramer, 1882; Loebker, 1887; Kuster; Vogt; Kraske; Popke; Volkmann; and more recently Broca and Hartman, 1897; Brin and Dujarier, 1898; Picqué, 1901; de Fourmestraus, 1906; and later, Gregoire, 1913).

According to Tavernier, Gregoire stresses the infrequency of loose bodies in the joint, and states that when present these appear to come from the anterior rim of the glenoid. He uses these arguments against the notch developing as a result of trauma or osteochondritis dissecans causing a portion of bone to become detached. He further argues that the dislocations are of too short duration for the notch to be caused by wear, and produces evidence from specimens of the neck of the humerus and from radiographs to show that the bone cortex and cancellous structure are entirely normal, so disposing of suggestions that the notch develops by crushing injuries. Gregoire concluded that the notch was congenital in origin, and he supported this contention by showing that the notch could exist in normal shoulders which had never been dislocated.

Tavernier himself describes the notch as the most enigmatic of the deformities.



A B

FIG. 41.—Radiographs of normal shoulder-joints in life, taken in maximum external rotation. Note the failure in one case (A), to show the head of the humerus and greater tuberosity in full profile. The angle here recorded is 148°, but is, of course, inaccurate in view of the position. In the other case (B) a good profile is obtained, although the case had never been subjected to a dislocation of the shoulder. The inclination of the axis of the head to that of the shaft is at an angle of 130°.

The Hatchet Deformity.—The specimen was X-rayed with the humerus in marked external rotation, and a normal control specimen was similarly treated. These are illustrated along with two living shoulders in maximum external

FIG. 42.—Radiograph of specimen of shoulder subject to recurrent dislocations, taken in external rotation. In this position the axis of the head of the humerus is inclined to that of the shaft at an angle of 139°.

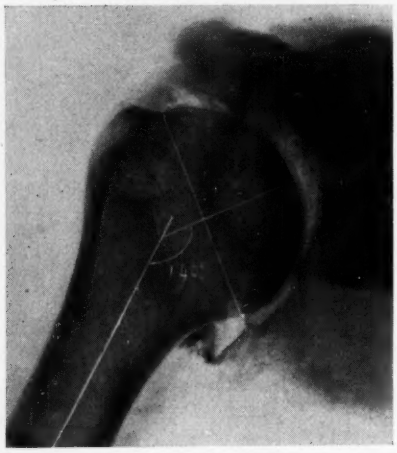


FIG. 43.—Radiograph of control specimen of a normal shoulder-joint taken in external rotation (some lipiodol is still present). In this position the axis of the head is inclined to that of the shaft at an angle of 140°.

rotation (*Figs.* 41–43), and show that in life the maximum external rotation does not always allow of the full profile of the greater tuberosity and head being shown, while both specimens show this profile. There is no essential difference

FIG. 44.—Radiograph of a normal shoulder in life, taken from above, in position of mid-rotation and with arm in the coronal plane. Note normal forward projection of the anterior lip of the glenoid. Note also the comparatively slight displacement of the head of the humerus needed to produce a direct anterior dislocation.

to be detected between the views of the head of the humerus in the case subject to recurrent dislocation and in the normal control.

Tavernier quotes Alibert as having analysed the hatchet deformity of the head as follows :—

1. The production of 'humerus varus' by a reduction of the angle between the axis of the head and that of the shaft of the humerus.
2. Flattening and limitation of the articular surface.
3. The lengthening of the neck.

This last is stated to be the most important factor, as it produces a cam-like action with rotation of the humerus, an effect assisted by the 'humerus varus'. The cam-like action would cause the head of the humerus to push against the anterior capsule in external rotation and the posterior capsule in internal rotation.

In the case described in this paper, the angle subtended by the axis of the head to that of the shaft does not appear different from that in the control specimen, nor does the neck appear to be at all lengthened.

In connexion with the 'humerus varus', Tavernier states himself that this is very difficult to assess, even where one knows precisely the position in which the radiograph was taken.

CONCLUSIONS

This case illustrates the detachment of the capsule from the rim of the glenoid cavity and its complete failure to become re-attached. This lesion is still not universally accepted because the majority of operations performed for the condition fail to expose the affected region of the joint. No other lesion has, however, been reported as consistently as has this lesion, by those who expose the anterior margin of the glenoid cavity (Bankart).

The posterior notch has not been described to anything like the same extent, but Continental surgeons, and particularly the French, seem to have laid much stress upon its presence. It will be realized how difficult it is to expose the notch at operation and it is a further example of how

the morbid anatomy is obscured by a limited surgical approach. Radiology, however, illustrates it well (*Fig.* 40). A view of the shoulder-joint from above, taken with the arm in the coronal plane and in mid-rotation, shows the presence or absence of the notch (*Fig.* 44). It is felt that this view should be taken in every case of recurrent dislocation of the shoulder.

The appearances in *Fig.* 40 suggest an absorption of not only the head of the humerus (the notch), but also of the anterior lip of the glenoid, when compared with the normal appearances as seen in *Figs.* 44 and 36. In the specimen the surfaces of the notch and of the anterior lip of the glenoid consisted of smooth, bare bone, and appeared perfectly adapted to one another. Erosion of the cartilage and absorption of the bone from recurrent trauma would seem to be the cause of the notch in this case. Over years, the trauma of dislocation must have a marked effect, and the increasing depth of the notch and the reduction in size of the anterior lip of the glenoid would account for the increasing ease and frequency of dislocation. This increasing ease of dislocation has not been accounted for by capsular detachment, which might usually be expected to be maximal at the outset.

These are the conclusions arrived at by a study of the specimens in this case, but the conclusions of Gregoire, as quoted by Tavernier, are different and are in favour of a congenital origin of the posterior notch.

Other than this pronounced posterior notch, no abnormalities of the head of the humerus were noted.

This case does not provide any evidence for or against Bankart's contention that the detachment of the anterior capsule is produced by direct anterior dislocation. The hypothesis is attractive, and it could be suggested that the shoulder might well mimic the hip, to the extent that there was a direct anterior dislocation of the shoulder similar to the direct posterior dislocation of the hip. In the latter the rim of the acetabulum, in the former the capsule and glenoid labrum, were detached. In both, the direct lesion alone was commonly associated with recurrence.

SUMMARY

The morbid anatomy of a case of recurrent dislocation of the shoulder is described and illustrated.

The detachment of the capsule and glenoid labrum from the rim of the glenoid, and a large posterior notch in the head of the humerus with reduction in size of the anterior lip of the glenoid cavity, were noted.

No loose bodies were found in the joint, nor were any other abnormalities of the head of the humerus disclosed.

It was concluded that the posterior notch had developed from erosion and absorption, the result of intermittent pressure, and that similar changes had occurred in the anterior lip of the glenoid cavity. These changes in this case would have been occurring over a period of nine years. The perfect adaptation of the surface of the notch to that of the anterior lip of the glenoid, both of which surfaces consisted of smooth, bare bone, was thought to be irrefutable evidence in support of this view.

The development of the notch on the posterior surface of the head, and the absorption of the anterior lip of the glenoid, would account for the increasing frequency and ease of dislocation, which is an invariable feature in cases of recurrent dislocation of the shoulder.

A radiograph of the shoulder, taken from above, with the arm in the coronal plane and in mid-rotation, is considered to be necessary in all cases of recurrent dislocation of the shoulder. This view will show a notch in the head of the humerus.

Bankart's theory of recurrent dislocation is discussed.

I would like to express my gratitude to Dr. Tavernier, of Lyons, from whose article I have quoted so liberally. I would also like to record my appreciation of the facilities put at my disposal by Dr. A. D. Fraser at the Bristol Royal Infirmary, and Dr. A. L. Taylor at the Bristol General Hospital, in their respective pathological departments, and for the help of the staff of the Department of Anatomy of the University of Bristol.

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BLAST EFFECTS IN WARFARE*

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It would probably be true to say that the violent disturbances, or blast, set up by explosions have only aroused real interest in our profession since the somewhat exaggerated accounts of the Spanish Civil War; but there has been a considerable amount written on the subject since the beginning of this war, and we are indebted to Professor Zuckerman and his collaborators for much interesting research on animals, and also to Sir Joseph Barcroft, who has been responsible for a large amount of work in connexion with the protection of human beings.

Fallon,[1] in his article in the British Journal of Surgery in 1940, gave a most interesting account of lung damage with the "intact thorax" due to the ordinary mishaps of life, and his paper naturally leads on to the study of the injuries to the lungs caused by explosions.

There is little mention of the effects of blast in the official medical history[2] of the last war, and this seems surprising in view of the very large number of casualties sustained on both sides. It was, however, recognized that corpses were found on the battlefield in the attitude of life with no external marks of violence whatever. Unfortunately practically no post-mortem examinations were made, and in any that were held the chief interest seems to have been taken in the lesions of the brain and spinal cord, where petechial hæmorrhages were observed.

Bowlby,[3] in his Bradshaw lecture in 1915, stated that he had only come across one case, and at the autopsy once again these petechial hæmorrhages in the brain and cord were noted, but no mention was made of the effect on the lungs or other viscera. However, Crile[4] at Rouen in 1917 carried out experiments to investigate the effects of explosions on rabbits in confined spaces. He observed that massive hæmorrhages were produced in the lungs, and at about the same time Maire and Durante noticed the effects on the brain and spinal cord. It was largely held at this time that the lesions were due to a negative pressure.

I have had the good fortune to discover in the archives of the Naval Gunnery School at Portsmouth a letter dated Aug. 31, 1918, written by Professor Leonard Hill, in which he demonstrates that he at least at that time appreciated these blast effects. He had carried out experiments on pigs, and he noted then that one of these animals died a day or two after exposure to explosion and that at the post-mortem there was both rupture of the alveoli and hæmorrhages in the lungs in scattered patches as though blast pressure had been exerted unequally. He had conducted experiments on frogs and observed that rapid raising of air pressure to 20 atmospheres caused no upset in the capillary circulation in the lungs, but this, of course, was not an instantaneous pressure rise.

He went on to say that the air in the alveoli may be compressed by blast acting on the abdomen —and this appears to be a possible explanation of the lesions found in the phrenico-costal sinus in human beings described by Osborn[5] and others. It would also explain the inspiratory position of the chest noticed by Allison[6] and others, and the rib markings found in some cases on the lung surfaces, suggesting that the lung is forced from below outwards against the chest wall.

I propose later to describe some cases where internal injuries were sustained by men in water in the vicinity of an explosion. One interesting point about these cases is that the lesions must have been produced by a pure hydrostatic pressure wave, observing that water to all intents and purposes is incompressible. Nevertheless, these patients must be considered to be blast cases in precisely the same way as if they had been in air at the time of their injury. Such cases were described in the *Journal of the Royal Naval Medical Service* in January, 1917, without apparent recognition of the nature of the lesions.

Most of the modern literature on the subject of blast concentrates on damage produced in the lungs, but later, injuries produced in other parts of the body will be briefly described.

THE NATURE OF BLAST

I take as the basis of my remarks work conducted by the naval experimental authorities over many years, though naturally their interest has been centred on blast effects with gunfire. Although gunfire and bomb explosions are not strictly comparable, the pressure effects produced by both must be reasonably similar. What, therefore, is the nature of the physical disturbances in air which we call 'blast'? It can be demonstrated that the physical effects are complex, as blast is not a mere mass movement of air or other gas. Through the courtesy of Professor G. I. Finch[7] a picture illustrating the complexity of the blast wave is produced. This is a photograph of the explosion of a mixture of pentane and air, and the actual exposure was $\frac{1}{1,000,000}$ of a second (*Fig.* 45).

* A Hunterian Lecture.

It will be observed that the gases ejected from the muzzle of the tube do not present a regular front; in fact, they appear as a molecular spray, but at the same time there is a preceding regular-fronted pressure wave demonstrated.

The velocity of the molecular spray is in the region of 5000 feet per second, that is considerably faster than sound in air, and the pressure wave is set up by the compression effect of the spray or windage wave acting against the inherent inertia of any substance—in this instance, air.

It can be accepted within reasonable limits that the force of both the pressure and windage wave will decrease proportionately to the square of the distance from the centre of the explosion. At the same time it is presumed that these waves are free to travel in air without encountering resistance. Should, however, an obstruction be placed in the path of the compound wave an exalted pressure will be built up, because the wave will be reflected from that obstruction and the pressure thus doubled. The speed at which the pressure wave travels is rapidly reduced, and it is in the region of the velocity of sound (1100 feet per second) at quite short ranges from the actual explosion, although the initial velocity may be three times greater—thus 11 ft. away from an explosion of a 55-lb. charge, the velocity was found to be only 2000 feet per second.

The time taken for the rise in pressure from that of the atmosphere to the blast peak is between $\frac{1}{2000}$ and $\frac{1}{10,000}$ of a second, and the positive pressure wave in air is, of course, succeeded by a negative wave of much longer duration.

It may be of interest to recount some results of research into blast effects by naval experimental authorities. This work has been done on the pressure effects produced by gunfire and therefore is not exactly comparable with those produced by the explosions of bombs, inasmuch as gunfire is a controlled form of explosion and the explosive used must not be a detonator. In the case of explosives used in bombs there is no limit to the rapidity of explosion compatible with the safe transport by aircraft of the bombs themselves, hence substances such as trinitro-toluol can be used in a bomb, whereas used as a propellant explosive they would almost certainly cause gunburst. At the same time it must be appreciated that because of the preliminary confinement of gasses generated by explosion in gunfire there will necessarily be a higher compressive effect than can possibly occur where a thin-walled bomb explodes.

It is admitted that a 'higher' explosive can be used in a bomb than in the charge behind a shell. Although the energy absorbed in fragmenting the thin bomb casing is relatively small, the energy generated by the exploding bomb is dissipated in all directions, but in gunfire the lines of force, excepting those applied radially to the wall of the explosion chamber of the gun, are primarily in the direction of the gun barrel. Hence, it may fairly be presumed that the total blast force at the muzzle of a gun is at least similar to, if not greater than, that of an exploding bomb of a similar weight of charge.

Zuckerman,[8] in his paper before the Royal Society of Medicine (January, 1941), states that the estimated pressure produced by an exploding bomb is between 100 and 650 tons to the square inch, but nothing like these phenomenal pressures

FIG. 45.—Photograph (exposure 1–1,000,000 sec.) of the explosion of a mixture of pentane and air. Note the irregular spray of gases issuing from the tube and the preceding regular-fronted pressure wave.

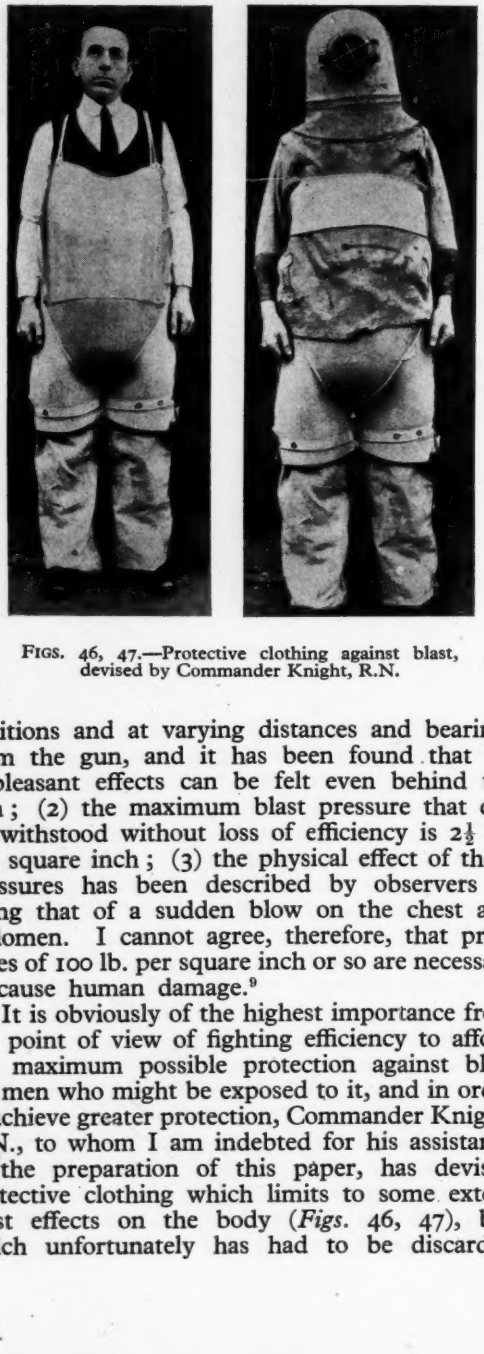
have been observed in gunfire. Pressure measurements with guns of any calibre have registered a maximum of 5 to 6 tons to the square inch at the muzzle with 25 tons to the square inch in the explosion chamber, and it has been proved that these pressures are very rapidly lowered within short distances of the gun itself. It should be noted that the blast effect of a gun is not wholly forward.

Although I may not state the instruments used in measuring pressure inside a gun, it might be of interest to describe how the conclusions of the effect of gun-blast have been arrived at.

Let us presume a loaded gun is being fired. At varying distances from this gun, starting close to the breech-end and disposed on a parabola with its axes centred at the muzzle, volunteer

observers are placed. Each man will have with him a pressure-recording apparatus to be described later. A round is then fired, and each observer is then questioned as to the physical effect it had on him. In order to try to eliminate the human element, the positions of the observers are then interchanged and a further round fired. This is repeated as necessary, and then the observations of these men are correlated together with the observed pressures at given points, and an estimate is arrived at of the safe area from the gun itself.

What I have just described is done over and over again with observers posted at various



FIGS. 46, 47.—Protective clothing against blast, devised by Commander Knight, R.N.

positions and at varying distances and bearings from the gun, and it has been found that (1) unpleasant effects can be felt even behind the gun; (2) the maximum blast pressure that can be withstood without loss of efficiency is 2½ lb. per square inch; (3) the physical effect of these pressures has been described by observers as being that of a sudden blow on the chest and abdomen. I cannot agree, therefore, that pressures of 100 lb. per square inch or so are necessary to cause human damage.[9]

It is obviously of the highest importance from the point of view of fighting efficiency to afford the maximum possible protection against blast for men who might be exposed to it, and in order to achieve greater protection, Commander Knight, R.N., to whom I am indebted for his assistance in the preparation of this paper, has devised protective clothing which limits to some extent blast effects on the body (*Figs.* 46, 47), but which unfortunately has had to be discarded because of the impracticability due to its weight, discomfort, etc. Commander Knight has subjected himself and others in this form of modern armour to higher blast pressures than could possibly have been withstood without it.

In consists of $\frac{1}{16}$-in. gauge aluminium, lined on its inner surface by 1-in. thick sorbo rubber. It will be seen that it covers most of the trunk; further, what is known as a shallow-water diving dress is worn over the head, chest, and arms. This shallow-water diving dress is a metal helmet with a short rubber suit extending to the abdomen and enclosing the arms. Thus to some extent a fairly rigid covering is provided for the whole body except the arms and legs.

In such 'knight's armour' this officer has approached the muzzles of guns in use on various bearings and at various distances. He has found that even with this protection the maximum blast pressure which he could endure (and this does not mean retaining efficiency) was 7½ lb. to the square inch. At this pressure he realized that he was within the danger area, and he described the effect as being "as though his bladder had burst".

These observations, made under exceptional circumstances with a good deal of protection, must demonstrate that a man exposed without such protection would have been liable to damage.

Further to illustrate this point I would like to mention some observations made on the bridge of one of H.M. ships some time ago. It was thought that when the very large guns of this ship were fired on an extreme after-bearing, that is, trained as far astern as possible, and with maximum elevation so that their muzzles were pointing towards the top of the bridge, such pressures would be experienced on the bridge itself that efficiency at least, if not life, would be impossible.

Once again Commander Knight was responsible with a co-observer for the personal investigation of this problem. The guns were trained into the position described, which meant that their muzzles were 23 ft. away from him, bearing 90°, that is, he could look immediately across the muzzle, but could not see down the bore. The guns were then fired and in these circumstances a pressure of 6 lb. per square inch was registered on the bridge, and this was sufficient to lift this officer off his feet and blow him back against the after-bridge screen, surely a very severe trauma for such a low pressure.

It may be of further interest to note that on the outer side of the bridge pressures of 20 to 30 lb. were registered; thus the interposition of the bridge screen, that is the metal wall of the bridge, was enough to afford a great measure of protection, but under these conditions this bridge screen was completely interposed between the observer and the gun.

In order to minimize the unpleasant effects of gunfire on the bridge of this ship, small metal baffle plates were fitted at an angle of 45° round

the top of the bridge itself and proved efficient, but at the same time it is extraordinary to note that these baffle plates were only efficient provided that no part of the body was exposed in the direct line of blast. They did not deflect the blast wave upwards as would be expected with a pure wind.

A further observation on the subject of gun-blast may be of value to illustrate the very rapid dispersal of the pressure wave in air. It has been found that 40 ft. away, at an angle of 45° to the line of the barrel of a gun at its muzzle, a pressure of only 2½ lb. to the square inch is set up, though at the muzzle itself the pressure is 5 to 8 tons.

To describe the method used in measuring these low blast pressures: a series of cells is prepared, each consisting of a metal box with a circular hole in one side. Inside these boxes are placed balloons inflated to various pressures. Interposed between the balloon and the hole is a thin aluminium plate and on the outer side is secured a sheet of tinfoil. Thus let us suppose one cell contains a balloon inflated to a pressure of 2 lb. per square inch—a greater pressure would cause collapse to some extent of the balloon and therefore inward movement of the aluminium plate and the tinfoil and necessarily an impression of the hole on the foil.

Similarly, suppose the pressure to be less than 2 lb.; there would be no collapse of the balloon, no inward movement of the plate, and no impression on the foil. So it will be seen that if a series of these cells is prepared and subjected to pressure, such pressure can be measured with a fair degree of accuracy. At the same time it should be noted that there will be some difference observed depending on the presentation of the meter—should it be facing the line of blast, higher pressures will be recorded than if the line passed across its face. Obviously, so simple an apparatus can be used to determine only low pressure, not above 50 lb. to the square inch, and higher pressures are measured by the complicated piezo-electric meter.

DISCUSSION OF CAUSATION OF BLAST INJURIES

There being three components in the compound blast wave in air, it is of importance to determine which is the traumatic factor and how it affects the body.

Zuckerman's[8] experiments on animals exposed to blast seem to demonstrate that the positive pressure wave is the cause of damage to the lungs, and that it acts by directly compressing the chest wall. At the same time the pressure wave acting on the abdomen and diaphragm must also have a considerable effect on the lungs, and experiments carried out on animals in water—to be described later—seem to confirm this.

I do not think we can entirely neglect the effect of the negative air suction wave. This wave has a great amplitude, as it starts at the peak of the positive pressure and descends relatively slowly from that point to below atmospheric pressure.

Daly[10] subjected goats to sudden decreases in air pressure and produced lesions in the lungs. Hence it is more than possible that the negative phase of the blast wave is injurious to human lungs, perhaps acting on lung tissue already damaged by the positive phase.

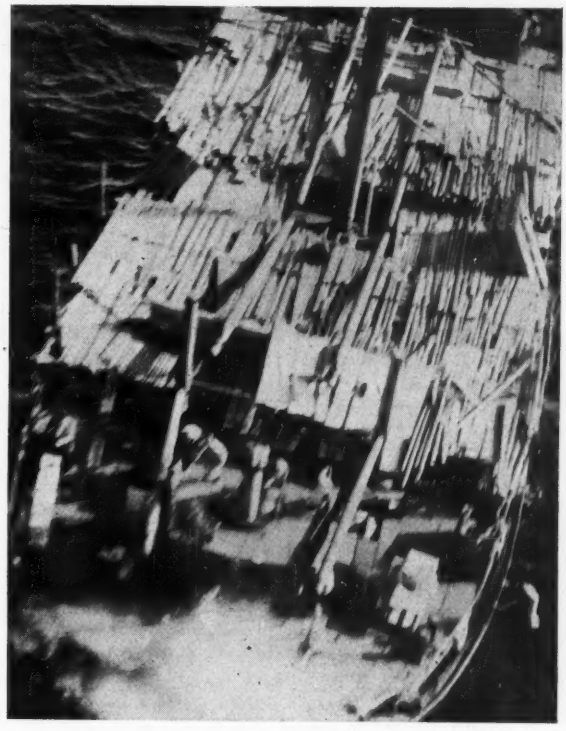


FIG. 48.—A seaman being blown over the side of his ship by the windage effect of the explosion of a nearby bomb.

The third blast component of mass movement of air or gas is capable of inflicting injury of any degree of severity and is responsible for the shattering effects produced in the immediate vicinity of an explosion. It should be noted that in water, where mass movement is of a small degree, even in close proximity to an explosion, these shattering effects are not seen.

Fig. 48 shows a man being blown over the side of his ship by the explosion of a nearby bomb. It will be observed that he is in an attitude of flexion, the normal response to fear, but in this case due to the 'folding up' effect of the explosion. The pressure wave must have passed him, of course, but the force which has lifted this man from the deck of his ship and flung him over the side is windage and not the pure pressure wave. It is quite evident, therefore, that contusional effects can be produced by the rush of gas from an explosion irrespective of any

damage which the pure pressure wave may have caused.

When we come to deal with blast effects in water, the problem is much simpler, as there is only a single pulse of pressure to be considered, the reflected wave from the sea bottom being of little importance except in very shallow water, where it would practically coincide with the primary wave—that is, of course, assuming that the exposed individual is on the surface.

The lesions caused in water, therefore, are due to a single high-pressure wave, and there is no question of a wave descending the trachea, or an after-suction wave. In brief, the effects on the body of explosions, both in air and water, are primarily due to the externally applied pressure wave, but in air both the windage factor and the after-suction waves play their part.

BLAST IN WATER

The effect of an explosion in water, as I have already pointed out, is not quite the same as in air, inasmuch as the primary pressure wave is not, except in the immediate vicinity, followed by a wave of mass movement, nor, to all intents and purposes, is there a suction wave.

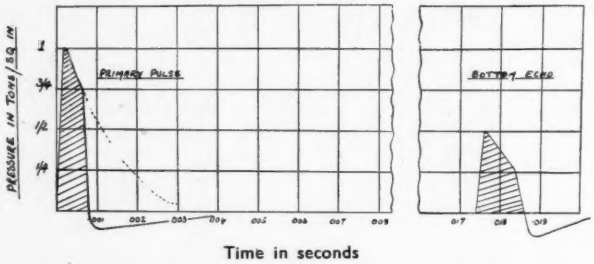


FIG. 49.—Diagram of blast pressure wave in water. The 'cut-off' effect in the primary wave is due to the neutralizing effect of the reflection of preceding waves reaching the surface.

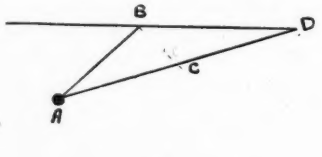


FIG. 50.—BD represents the surface of the water. The explosion occurs at A. AB and AD are primary waves and BC the reflected wave from the point B at the surface.

The pressure wave in water travels at the same speed as it does at first in air, i.e., 5000 ft. per second, and it should be noticed that this is the same as the speed of sound in water. The rate of travel is constant and does not diminish as the centre of explosion recedes.

The rate of fall of pressure is much slower than in air, and varies directly as the distance. Thus, suppose that 60 ft. away from an explosion the pressure was 1 ton per square inch, at 120 ft. it would be ½ ton, at 240 ft. ¼ ton, and so on; hence it will be seen that pressure effects will be experienced over a much greater distance in water than in air for the explosion of a given charge. The thickness of the pressure belt is estimated to be about 6 in.

When we come to deal with the pressures set up by an explosion at some depth in water a new and important factor arises. When the pressure wave reaches the surface there is no transmission of the pulse to air, but there is reflection as a wave of tension at an angle equal to the angle of incidence, just as an oblique beam of light would be reflected from a plane mirror, and this reflected pressure wave will have to some extent a neutralizing effect on the positive waves reaching the surface at a greater distance from the explosion centre (*Fig.* 49).

Consider an explosion to occur at a point A below the surface of water (*Fig* 50). Pressure waves will be set up and some will travel to the surface, where reflection will occur. Imagine AB to be the path of the primary wave reflected at point B along line BC. The reflected wave BC will have a neutralizing effect on the wave AD which reaches the surface at the more distant point D, but because the sum of AB and BC is greater than AC, the reflected wave BC will only exert its effect after the peak of the primary wave AD has passed, hence the effect will be to cut short the duration of the pressure wave, but not to affect its amplitude.

Of course, in addition to surface reflection there will be reflection on any dense object, such as the bottom or sides of a tank, but in these cases the reflection is positive and a doubled pressure is built up. Should, however, the obstruction be of low density compared with water and therefore presumably floating, the pressure wave will not be exalted by reflection at the surface of that object, but will change from a static pulse into a wave of kinetic energy and the object will be projected into the air above. This effect is seen at the surface of water over an explosion, where the superficial layer disintegrates into a dome of spray because of the change from static to kinetic energy. This dome of spray is of no great height and is visible an appreciable time before the much higher column of water due to escaping gases appears.

Now consider the human body, which has roughly the same density as water. When the pressure wave impinges on the body there will be no reflection, but the pulse will be transmitted through the tissues without displacement, just as if the body were so much water. However, when the transmitted pulse encounters an air cavity in the body, e.g., the lungs, the same phenomenon will occu as has been described

above, that is, the static wave of pressure will change into a wave of kinetic energy in the layers of tissue lining that cavity and a disruptive effect will occur. Hence it can be understood that the lungs and other gas-filled cavities in the body are particularly susceptible to damage from the pressure wave, even though the body itself may not be deeply immersed.

Should a man be wearing an inflated rubber jacket when exposed to an explosion in water, he will be afforded considerable protection, as the gas-filled jacket will largely absorb the disruptive effect of the change to kinetic energy of the pressure wave.

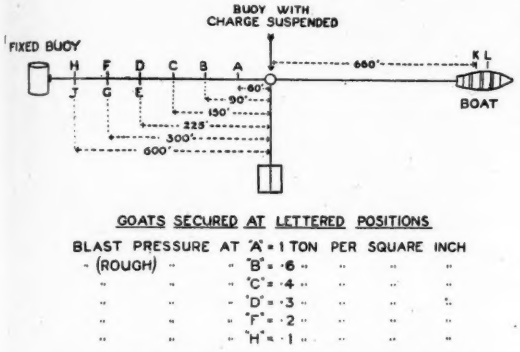

BUOY WITH CHARGE SUSPENDED
FIXED BUOY
H F D C B A
J G E
60'
90'
150'
225'
300'
600'
660'
K L
BOAT
GOATS SECURED AT LETTERED POSITIONS
BLAST PRESSURE AT 'A' = 1 TON PER SQUARE INCH
(ROUGH) " " 'B' = ·6 " " " "
" " " 'C' = ·4 " " " "
" " " 'D' = ·3 " " " "
" " " 'F' = ·2 " " " "
" " " 'H' = ·1 " " " "

FIG. 51.—Diagram illustrating the first experiment (*see text*).

An experimental study of blast effects in water was carried out off Spithead early in September last in conjunction with the Porton Experimental Authorities and H.M.S. *Vernon*. For the purpose of this experiment goats were used and subjected to the explosion of a 300 lb. charge in water of a depth of 90 ft.

It was, of course, essential to choose a calm day to ensure that the immersed goats did not inhale any seawater. The animals were buoyed up by inflated rubber tyres round their necks, and were secured to a line at various intervals. One end of the line was secured to a fixed buoy, and 600 ft. away the other end was made fast to another buoy, from which was suspended at a depth of 8 fathoms (45 ft.) a charge of 300 lb. of T.N.T. (*Fig.* 51).

The boat with the operators and spectators then retired 220 yards and two further control goats were put over the side and the charge was fired electrically.

As soon as the upheaval had subsided, the boat picked up the goats, and it was astonishing to find that only the one nearest the charge (goat A) was dead. Goats at positions B, C, D, F, and H were killed with a humane killer, and it was noticed that the survivors had considerable difficulty in standing for a short time, but soon recovered, this being in accordance with observed human effects.

It is proposed only to refer to the more obvious post-mortem results of this and the next experiment, as the findings, both macroscopical and microscopical are fully described in the article by Professor Cameron and others on p. 49.

No blood was seen at the mouth or nostrils of any of the animals, and there was no external evidence of injury, but the temperatures of those longest immersed had dropped 5° F.

On completion of the experiment the party returned to R.N. Hospital, Haslar, where post-mortem examinations were conducted by Captain Short, R.A.M.C., and Captain Blount, R.A.V.C. In each case lung effects were found of diminishing severity with increase of the distance from

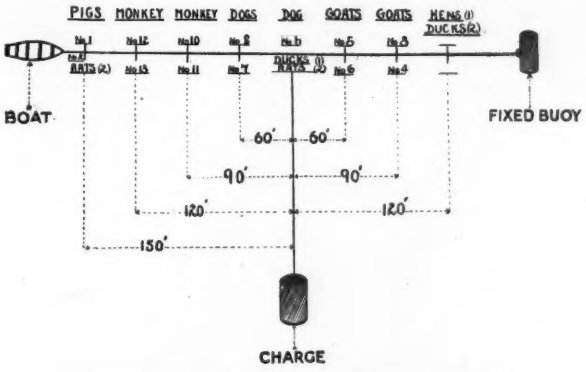

PIGS MONKEY MONKEY DOGS DOG GOATS GOATS HENS (1) DUCKS (2)
No. 1 No. 12 No. 10 No. 8 No. 6 No. 5 No. 3
RATS (2) No. 13 No. 11 No. 7 DUCKS (1) RATS (2) No. 9 No. 4
BOAT
FIXED BUOY
60' 60'
90' 90'
120' 120'
150'
CHARGE

FIG. 52.—Illustrating diagrammatically the second experiment (*see text*).

the explosion, but it is surprising to note that in no case was there intra-abdominal damage.

The lung lesions were similar to those found by Zuckerman in his experimental animals and also in fatal human cases. The four goats nearest to the explosion presented rib markings on the costal surfaces. All lung specimens showed hæmorrhages on the phrenico-costal margins, and there were more or less extensive hæmorrhages into the substances of each, more marked in the right lungs than the left. Goat F showed multiple small hæmorrhages on the pleural surfaces of both lungs.

The surviving goats were returned to Porton and kept under observation, but none showed any ill effects.

The object of this experiment was to determine the blast effect in water, and it appears that, with goats, pressures of a fairly low order are sufficient to cause lung damage, but that the pressures must be very much higher to effect intra-abdominal injury; further, that phrenico-costal margin lesions in the lungs do not indicate the probability of tears in the liver or spleen as suggested by Osborn.[5]

A further experiment was done towards the end of September under the same conditions of depth of water and charge, though the sea was rougher, but this time a variety of animals was used and some of them received partial protection by means of Sorbo rubber shields. The animals were arranged as in *Fig.* 52.

The charge was fired and the animals were rapidly picked up. It was found that some of the large animals were dead, and all survivors were killed with a humane killer. Those that were alive, as in the last experiment, could not stand when first hoisted out of the water.

Post-mortem examinations were carried out at R.N. Hospital, Haslar, by Professor Cameron and Captain Blount, R.A.V.C. The main points of interest observed were: The animals secured directly over the charge were not "blown to pieces", though they suffered severe damage to lungs and abdominal viscera, but the duck was in fact disintegrated, and this is thought to be due to the fact that it would be riding much higher out of the water and would thus be forced up into the air by the pressure wave changing into a wave of kinetic energy at the surface of the water.

The chest of dog No. 8 was protected by a 1-in thick Sorbo rubber jacket, but it suffered much more severe and extensive lung damage than did dog No. 7, which had no chest protection, but whose abdomen was covered with similar Sorbo rubber. This latter dog when recovered from the water seemed to be in relatively good condition, though partially paralysed in the hind legs, whereas dog No. 8 was dead (*Figs.* 53, 54).

A similar state of affairs was found in goats Nos. 5 and 6, which were exposed at the same distance. No. 6 had chest protection and suffered more severe lung damage than did No. 5, whose abdomen was protected.

Abdominal lesions were found in dog No. 6, which was immediately over the explosion. This animal's stomach and liver were extensively lacerated, and there was a small tear in the upper pole of the left kidney. There was extensive damage to the lungs, which were torn on removal from the chest owing to old adhesions to the chest wall.

Abdominal damage was found in the monkeys, especially No. 13 (*Fig.* 55). There was no actual laceration, but the lesions seen were either small subperitoneal hæmorrhages or hæmorrhages, some quite extensive, into the mucous membrane of both large and small intestines. This is a most interesting finding, as in a surviving animal it would undoubtedly lead to melæna or perhaps late peritonitis or abscess formation, and would appear to be the cause of melæna seen in human blast cases.

It is of considerable practical importance to the understanding of the causation of blast injuries to note that in hens damage was observed in the lung tissue itself, but not in the prolongation of the air sacs into bone, where protection would be afforded. Further, that although the diaphragm of these birds is but a thin membrane, it was in no case torn.

The pigs showed minor lung damage, small hæmorrhages along the phrenico-costal margins being noticed, but no abdominal lesions.

It will be observed that blast effects occurred in the lungs of all these animals, whether single or multiple stomached, but that the animals whose

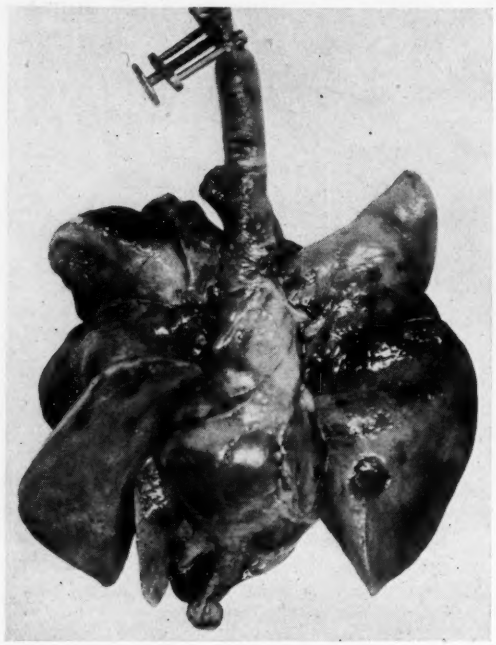


FIG. 53 (Dog No. 7).

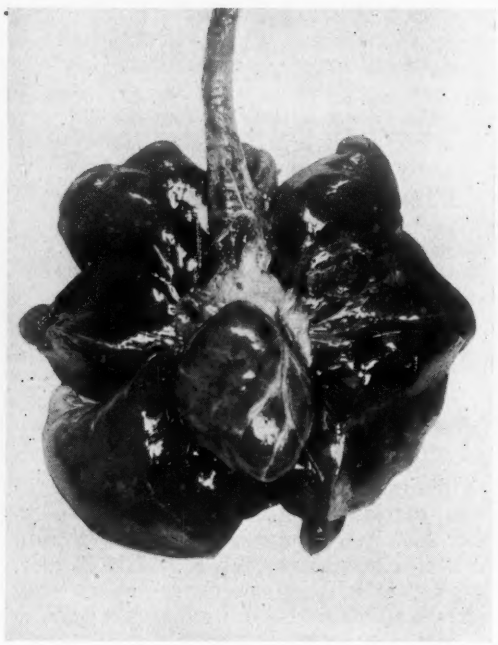


FIG. 54 (Dog No. 8).

FIGS. 53, 54.—Blast effect on dog's lungs. Note the very much more extensive hæmorrhages in No. 8 than in No. 7. The chest of No. 8, and the abdomen in No. 7, were protected.

abdomens were protected received less lung damage than those whose chests were covered. Thus it is fair to assume that although the pressure wave applied to the chest undoubtedly may cause damage, the upthrust applied to the diaphragm via the abdomen must be equally, if not more, dangerous. Another most significant

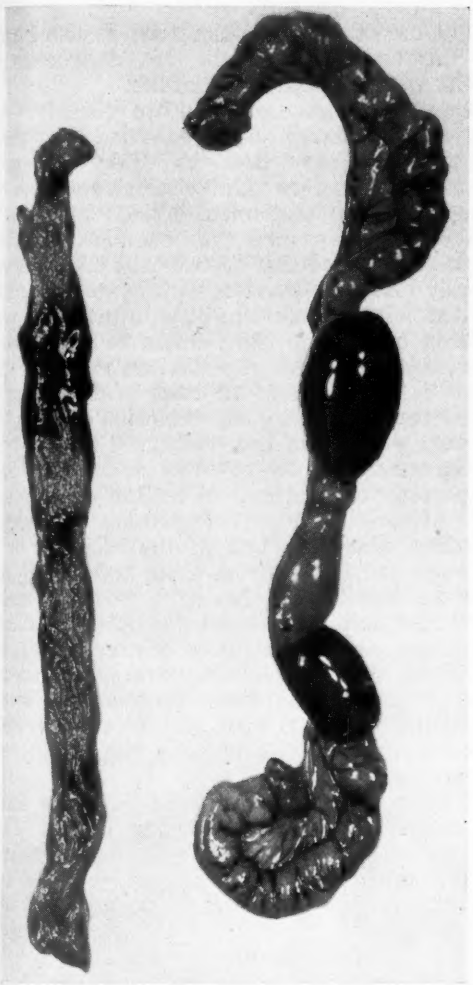


FIG. 55.—Blast effects in small intestine of a monkey, showing submucous and subperitoneal hæmorrhages.

finding is that damage was usually found in gas-filled cavities such as the stomach, intestine, and pulmonary alveoli, whereas no damage was found in fluid-filled cavities such as the renal pelvis, urinary bladder, and gall-bladder. This, I think, bears out the theory of causation of trauma which I have already discussed.

HUMAN BLAST INJURIES

I have endeavoured so far to demonstrate the nature of blast and to show that even low pressure suddenly applied to the body can cause damage.

Let me turn now to a consideration of the human injuries that may be caused by blast. It is of importance to note that internal blast injury without external marks of violence is rare —I have personal knowledge of only 4 such cases in roughly 1500 casualties—in practically every case there will be a coexisting wound of some kind or some other condition due to crushing, asphyxia, and so on.

Blast Lungs.—The patient will invariably be very shocked, and for an hour or two may show no other signs. He will presumably receive treatment for this condition and first-aid treatment for his wounds.

After a while pain in the chest and difficulty in breathing is complained of, and a short, rather grunting cough develops which obviously causes great distress.

The patient is pallid but somewhat cyanosed. His temperature will be subnormal, but the pulse and respiration rates will be considerably raised.

The cough is at first unproductive, but later sputum which is blood-stained, possibly heavily, is brought up.

A further symptom noticed in some cases, thought not in mine, is generalized pain in the trunk, thought to be due to multiple small intramuscular or paraneural hæmorrhages.

Physical examination of the case at an early stage may reveal very little. At first the only thing noted may be limitation of the respiratory excursion; later on scattered rales will be found on auscultation, and still later, if the patient survives so long, evidence of patchy consolidation of the lungs and possibly signs of a pleural effusion may be observed.

A typical early radiograph of the chest will show multiple small scattered shadows throughout the lungs (*Fig.* 56).

An incident of interest in connexion with this condition occurred during the campaign in Norway. Seven men on board one of H.M. ships were in the vicinity of a magazine hatch when the ship blew up. These men managed to reach shore, but one of them felt so ill that he was unable to stand and had to be carried by his comrades to shelter in a school, where the party turned in for the night. The sick man was horrified to wake the next morning and see the remaining six men lying with rather blue faces and to be informed that they were all dead.

Undoubtedly these men had all suffered from "blast lung", and it is noteworthy that they were able to save themselves and even to help their shipmate to cover before collapsing. This is in accordance with observed experimental effects on animals (Zuckerman[8]), which are often able to run about for some time after exposure to explosion.

At my hospital we have had only 4 pure blast-lung cases and all these survived. They presented the typical clinical and radiological picture, the predominant symptoms in each case being dyspnœa and cough. In none of these cases was there very much blood in the sputum.

The conditions cleared up fairly rapidly and discharge from hospital took place 40 days, 21 days, 19 days, and 18 days after admission, respectively.

Six other diagnosed cases with severe complicating wounds all died.

I have described above what I consider is the typical clinical picture of a blast-lung case, but all sorts of variations can, of course, occur; however, the symptoms which should lead to suspicion of blast damage to the lungs are hæmoptysis (slight or severe), pain in the chest, and a short cough accompanied by dyspnœa.

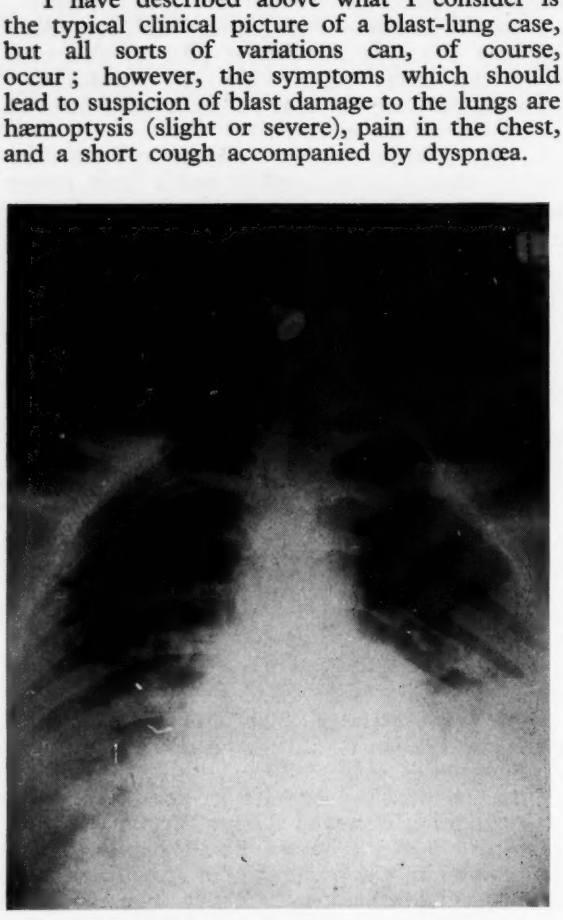


FIG. 56.—Radiograph of chest demonstrating blast lungs.

A point of high practical importance arises in complicated lung cases, and that is to what extent it is safe to give an anæsthetic. Undoubtedly inhalation anæsthetics must be avoided if possible, but there are many cases with severe wounds that demand an urgent surgical procedure at the earliest practical moment in order to save life, and I do not see that the surgeon of his anæsthetist should be deterred by the presence of a lung lesion. There must be few surgeons of any experience who have not at some time or another operated on a case of early acute pneumonia in mistake for one of acute abdominal lesion, and the patient usually survives the surgical attention. On this analogy I see no reason to be unduly worried by the risk of anæsthetic in a blast-lung case, providing the general condition is good enough.

The choice of anæsthetic, however, is strictly limited, and if the use of a local anæsthetic would not provide a wide enough surgical field, I consider there is only one suitable method, that is nitrous-oxide and oxygen, possibly preceded by a minimum dose of pentothal or evipan. In no circumstance should a spinal anæsthetic be given to such a case with a degree of shock and lowered blood-pressure.

The use of ether or chloroform is also barred and the barbiturates alone are dangerous on account of their depressant effects.

Blast Abdomen.—When we come to deal with this condition, a very varied picture is possible. We may have to deal with gross trauma to hollow or solid viscera, and, on the other hand, the abdominal lesions may be of such small importance that complete recovery in the course of a day or two will be observed. O'Reilly[11] had 5 such cases with acute abdominal pain following exposure to blast, of which 2 were operated on and no gross lesion found, and all recovered except one who died on the operating table. I have notes of 10 cases of men actually in the sea injured by an explosion during the last war, 5 of whom had melæna, 8 hæmoptysis, 2 hæmaturia, and all 10 acute abdominal pain, tenderness, and rigidity. All these cases recovered without surgical intervention. However, a patient who has been in the vicinity of an explosion and suffers from acute abdominal pain must be regarded as having a possible severe abdominal injury. It cannot be immediately ascertained whether this is so or not, but caution should be the surgeon's watchword in such a case. It can do no harm to treat for shock, meanwhile keeping the patient under observation, with due regard to a rising pulse-rate and increase of abdominal signs.

I have seen only one case of gross intra-abdominal damage due to blast in air. This man was admitted after an air-raid, the particular circumstances of the incident not being known. He was very shocked and practically unconscious. His abdominal physical signs were complete rigidity and generalized tenderness. There was no evidence whatever of any external injury. After blood transfusion, the abdomen was opened through a left paramedian incision, and on incising the peritoneum a large amount of free blood was seen and removed by suction. About 7 ft. of the jejunum showed blast effects, the peritoneal coat being torn. About 12 ft. from the duodenojejunal junction the small intestine was completely torn across and the omentum lacerated into shreds. A large retroperitoneal hæmatoma was discovered on the left side of the abdomen, and in two places the peritoneum covering the posterior abdominal wall was extensively split.

The ends of the torn jejunum were closed and a lateral anastomosis was performed. The torn peritoneum on the posterior abdominal wall was sutured and the abdomen was closed with

drainage. Two further small blood transfusions were given, and later a continuous drip intravenous glucose saline was started, but the patient survived for forty-eight hours only, his condition gradually deteriorating to death.

Other regions of the body which may be affected are, of course, the brain and spinal cord, and blast concussion will in no way differ from concussion caused by any other form of violence.

An interesting condition has been noticed in men who were in the sea when the depth charges in their sinking ship exploded. The sensation experienced has been described by the Medical Officer as of a violent blow in the back which caused a temporary paralysis of the legs. Several men were observed to cease their efforts to save themselves, some even being unable to continue holding on to flotsam.

The survivors from this ship all suffered from various degrees of abdominal pain. One officer developed severe melæna and died on the third day, and one rating developed general peritonitis and was successfully operated on six days after injury when the rescuing ship reached port.

Ears.—The fragile membrana tympani is naturally very frequently ruptured, and in many cases where severe lesions in other parts of the body are found, the ear-drums will have been damaged too.

Eyes.—The eyes may be affected either by foreign bodies such as dust and so on, or by the direct violence of the blast wave.

Fig. 57 is a photograph of a pure blast effect on the eye—the patient suffered no other trauma whatsoever. He was about 40 yd. away from an exploding bomb, facing towards it. It will be noticed that he has severe chemosis and that there is a small hæmorrhage in the anterior chamber. This patient complained of photophobia and a feeling as of grit in the eye. The hyphæmia absorbed quite easily, though if it had not it would have been necessary to remove the unabsorbed blood. A further case is worthy of description: A man was running for shelter when a bomb dropped in close proximity and he was blown over. Although shaken, he received no wound and got up and assisted in the extrication of some of his comrades from a damaged refuge. Shortly afterwards he noticed when trying to wink his right eye that he was blind in the left, but after three hours he could see quite clearly with both eyes. About one month later gradual deterioration of vision in the left eye set in, and when seen by the Ophthalmic Specialist at Haslar three months after the original injury, hand movements only could be detected by the affected eye. Ophthalmoscopic examination then revealed a large intra-ocular hæmorrhage occupying two-thirds of the vitreous and completely obscuring the optic disc. There was no increase of tension. This case showed no improvement and the man was eventually invalided from the Royal Navy.

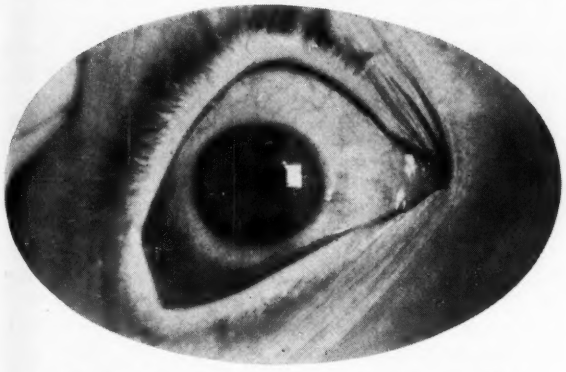


FIG. 57.—A case of blast trauma to the eye. Note the small hæmorrhages into the anterior chamber and chemosis.

Here, therefore, are two cases, one with a hæmorrhage into the anterior chamber and one into the posterior chamber of the eye. These did not present the usual clinical picture, and more commonly, simple chemosis only is noticed. Altogether 15 cases (1 per cent) of primary and secondary blast effects on the eye have been seen at my hospital.

So far we have had under consideration the clinical details of cases in which the pure primary blast wave has been the cause of damage, but I have already pointed out that such cases are few and far between. Of much more importance from a practical point of view are what I prefer to call the secondary effects, that is, injuries caused by flying bomb fragments, debris, etc., but these in themselves are outside the scope of this paper and are in the realms of pure war-time surgery. The other lesions which may be the result of an explosion, such as compression injuries due to falling masonry, asphyxia due to explosive gases or burst gas mains, are also outside my purpose here. I have restricted my remarks to those patients who present primary lesions, possibly complicated by other injuries.

We turn now to another result of blast injury. Wakeley and Rowlands[12] have described in a recent paper in *The Lancet* 3 cases of fat embolism one of which was directly referable to injuries sustained during an air-raid. This particular case was an officer who suffered from compound fractures of both tibiæ and fibulæ and who developed paralytic signs on the second day and died on the third. At necropsy multiple hæmorrhagic areas were found in the brain and spinal cord, which on microscopical examination proved to be fat emboli. The lungs showed evidence of consolidation, with extensive exudate into the alveoli consisting of fluid and red blood-cells. Numerous fat particles of various sizes could be seen scattered throughout the tissue, some in the pulmonary capillaries and arterioles and others in the alveoli. Here we have a case of death

after an explosion injury due to fat embolism associated with a bilateral fracture. At the same time, however, small quantities of fat may be found in the lungs in a high percentage of deaths due to any kind of trauma, and Professor Hadfield has pointed out that only when the quantity of fat is large and there is embolism of the glomerular capillaries can much importance be attached to the condition. It will be appreciated that any blow received by the body may cause damage, for example, to subcutaneous fat, and therefore autolysis, which is in itself liable to lead to fat embolism. This, however, will be a late rather than an immediate blast effect.

TREATMENT

The scope of this paper permits only suggestions for the treatment of those patients who have been subjected to explosions but present no external signs of violence—what I like to call 'primary' blast cases. Any case of blast trauma which requires hospital treatment will present symptoms of shock to a greater or less degree, and it is with this condition that we are primarily concerned. When the diagnosis of blast trauma can be made, the treatment will be on general principles, and as regards abdominal injuries the only point which I wish to emphasize is that most cases presenting symptoms recover without surgical intervention.

Let us now consider the blast-lung case. Here we are confronted with a difficulty; the patient will invariably suffer from a severe degree of shock, and therefore intravenous transfusion might be considered necessary, but both the clinical and pathological pictures of blast lesions of the lungs closely resemble those of acute pulmonary œdema. It would be wrong, therefore, to overload the venous circulation with extra fluid which could only lead to increased alveolar flooding and embarrassment of the right heart.

It has been suggested that hæmo-concentration may occur in the blast-lung case, with corresponding strain on the right heart and venous congestion. It may be thought necessary to perform venesection for the relief of this condition, but I consider this would be unpardonable until the patient has recovered from his stage of shock.

To my mind, the first form of active treatment should be continuous administration of oxygen, and in my experience these patients tolerate the old-fashioned nasal catheter much better than the B.L.B. bag, and this opinion has been confirmed by Professor Ryle.

The respiratory embarrassment and the distressing cough, both of which cause so much restlessness, call for the administration of some form of sedative, and I have found that heroin, in doses of $\frac{1}{8}$ gr., repeated as necessary, is most satisfactory and has not the depressant effect of morphia.

Another point to be considered is that these patients with damaged lungs will be particularly liable to air-borne infection, so it is of importance that the danger of droplet infection from surrounding nurses, anxious relatives, and so on, be minimized by the wearing of masks and strict attention to nursing hygiene. Further, these cases should not as far as possible be nursed in a general ward, but rather under conditions of isolation.

I am doubtful whether the administration of sulphanilamide or sulphapyridine is justifiable in the first twenty-four hours. On general grounds no doubt it would be a good thing to attempt to produce a high concentration in the blood at the time when pneumococcal infection is liable to occur, but these drugs are undoubtedly somewhat depressant and the fact that they often induce vomiting would only aggravate the danger of the condition.

It should not be necessary to lay stress on the fact that for such patients rest and quiet are essential.

CONCLUSION

I have endeavoured to describe in broad outline typical lesions produced in the human body by explosion blast and to put forward suggestions for treatment.

At some length descriptions have been given of experimental effects of blast in air and water, with particular reference to results obtained in animals subjected to underwater explosions.

To summarize :—

1. Even low pressures suddenly applied to the body can cause injury.

2. In air, the danger of primary blast trauma is not great owing to the very rapid decrease in the pressure-distance factor. Nevertheless, in the vicinity of rigid objects, for example the walls of a room, the pressure is built up by reflection and hence such situations are more dangerous.

3. In water the pressure phenomena are similar to those in air, but the pulse is transmitted and injury may be sustained over much greater distances than in air.

4. The explanation of physical damage in water is thought to be that a static wave of energy (pressure) changes into a moving form at surfaces in contact with gas, with consequent disruption. Although the matter has not been discussed, it is for consideration that a similar effect occurs in air, where, however, other factors such as the windage and after-suction waves have to be taken into account.

5. Experiments on animals to investigate results of underwater explosions have been described.

It is hoped, when weather conditions improve, to carry out further experiments with underwater explosions to investigate several points that have arisen in the course of the preliminary trials.

I am indebted to the Medical Director-General of the Navy, Surgeon Vice-Admiral F. Sheldon Dudley, F.R.S., for permission to publish this paper.

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PATHOLOGICAL CHANGES PRODUCED IN ANIMALS BY DEPTH CHARGES

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IN the middle of 1916 several cases of injury due to blast in water resulting from an exploding mine or depth charge were reported by Medical Officers in the Royal Navy. These cases all seemed to have a somewhat similar symptomatology—namely, hæmoptysis, bloody diarrhœa, and hæmaturia. Most of the cases experienced the sensation of a violent blow upon the immersed parts of the body and some became temporarily unconscious. Although the occurrence of such cases was known and commented upon in some of the medical journals, yet as soon as the war ended so did interest in this subject, as no cases were reported afterwards.

It was not until the present campaign that the real seriousness of water-blast injury became fully manifest. Reports have been coming in from medical officers at home and abroad, giving clinical data of naval patients who have suffered from water blast.

The present intensive warfare with repeated dive bombing and torpedo attacks on our ships has meant that quite often the majority of a ship's company is in the water after a ship has sunk. Should such an occasion arise and a depth charge, mine, or torpedo explode in the vicinity there is a grave danger of most of the men losing their lives.

The clinical manifestations of patients who have suffered from water blast vary from slight spinal concussion accompanied by bloody diarrhœa to laceration of the abdominal organs and lungs.

It has been realized that the mortality from water blast is very high and it is quite conceivable that many war casualties are due to this factor alone. More post-morten examinations on fatal cases are required, and it is hoped that facilities will be forthcoming whereby post-mortem examinations on fatal cases can be carried out on all possible fatal casualties occurring in the sea.

Moreover, medical officers should be informed that these examinations are important because valuable information may be forthcoming which will indicate what precaution or protection is necessary to prevent water blast from injuring persons in the water.

With the object of finding out the real causation and mechanism of these thoracic and abdominal lesions, and also to see if any form of simple protection could be devised, a series of animal experiments was carried out in September, 1941, at Spithead.

In the first trial goats were used and subjected to the explosion of a 320-lb. charge of T.N.T. suspended at 48 ft. in water of a depth of 90 ft. The experiment was arranged as shown in *Fig.* 51 in the preceding paper.

After the explosion the animals were rapidly picked up and only the goat at position A was found to be dead. Animals at positions B, C, D, F, and H were killed and subjected to post-mortem examination as soon as possible. None showed any external signs of violence, but most were temporarily paralysed. The surviving animals were kept under observation, but showed no late ill effects.

A further experiment was carried out later in September, using a variety of animals (dogs, monkeys, goats, etc.), some of which received

partial protection either to chest or abdomen by means of 1-in. thick sorbo rubber shields. The same conditions of charge and depth of water pertained, but this time the sea was rougher. The arrangement of the trial and the pressures set up can be seen in *Fig.* 52, also in the preceding paper.

Since the subject is there discussed at length by Surgeon Commander Williams, this report is confined to the pathological changes found in animals subjected to the experiments. Further work is continuing.

bleeding from orifices, though carefully looked for, was not seen, except in three animals showing epistaxis.

Respiratory System.—The most severe changes were found in the respiratory system and included: (1) pulmonary hæmorrhage, (2) bronchial and tracheal hæmorrhage, (3) acute vesicular and interstitial emphysema, (4) occasionally pneumothorax and hæmothorax.

Pulmonary hæmorrhage was as a rule most severe in animals nearest the depth charge (*Fig.* 58). It occurred in the form of bilateral diffuse

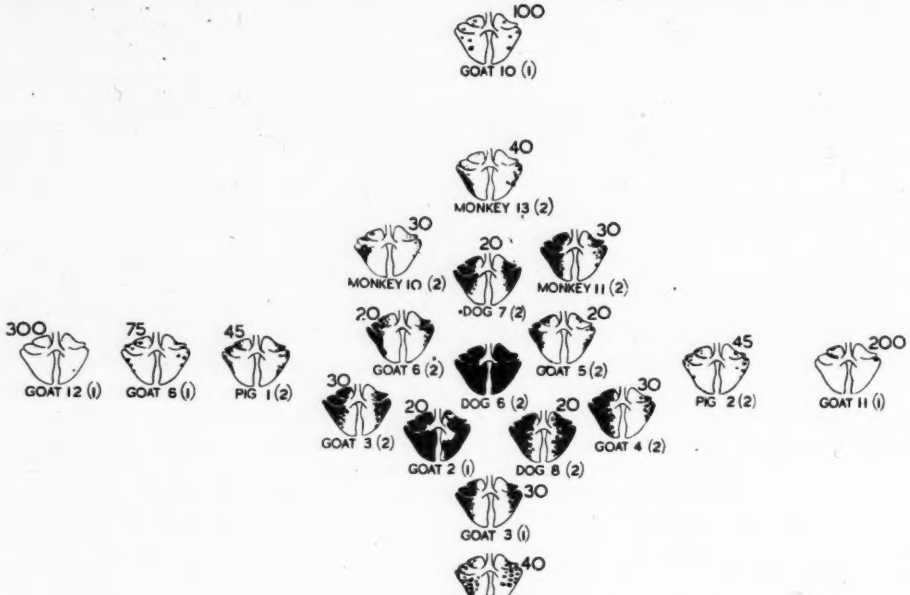

FIG. 58.—Showing the distribution of hæmorrhage in the lungs. Large numbers indicate distance from depth charge.

MORBID ANATOMY

Mortality-rates are shown in *Table I*, from which it appears that 84·6 per cent of animals within 40 yd. of the depth charge met with instant death. A striking feature was the infrequency of external evidence of injury. Bleeding from the nostrils was present in three animals only, dog 6 placed immediately over the depth charge, dog 8 20 yd. away, goat 3 30 yd. away. There were no lacerations or bruises in the skin, subcutaneous tissues, or muscles, even in the animals nearest the site of explosion. A fracture of the ilium and pubis was found in goat 7 100 yd. from the depth charge, but this may have been the result of a kick from another animal. No other fractures were found in the long bones, ribs, vertebral column, or skull. No injuries were found in the eyes or external genital organs; scarlet, almost black, areas involving the whole or the greater part of a lobe or lobes, sometimes with associated rib markings and invariably with scattered smaller hæmorrhages, 1 to 2 cm. diameter, and petechiæ. A detailed analysis of these lesions brings out a number of interesting points :—

1. The most severely affected animal was dog 6, placed immediately over the depth charge, its lungs being disintegrated in places, with massive hæmorrhage throughout all lobes. Massive hæmorrhage was seen also in goats at 20 and 30 yd. respectively, in a dog at 20 yd., and a monkey at 30 yd. (*Table II*). Lesions of the lungs tended to become much less severe after 30 yd.

2. Pulmonary hæmorrhage was, however, not necessarily an index of severity of injury. Goat 5 at 20 yd. was found dead, yet it showed slight

lung lesions. Monkey 10 at 30 yd. was *in extremis*, monkeys 12 and 13 were found dead, yet none of these showed striking lung hæmorrhage. In the goats lung lesions were directly proportional to the distance from the depth charge; in other species this was not so well defined.

Thus goat 2 (series 1) at 20 yd. was found dead and showed massive pulmonary hæmorrhage (*Fig.* 59); goats 5 and 6 (series 2) at 20 yd., both dead, showed moderate or severe hæmorrhage; goat 3 (series 1) at 50 yd., though alive, was severely injured; goats 3 and 4 (series 2) at 30 yd., both dead, had extensive pulmonary hæmorrhage (*Fig.* 60); goat 4 (series 1) at 50 yd. showed moderate changes; goats 5, 7, and 10 at 75 yd., 100 yd., and 100 yd. respectively, had slight lesions diminishing in severity.

3. The hæmorrhage resembled infarction of lung tissue in 9 of the 21 animals, i.e., 43 per cent. This very severe change was confined to animals within a radius of 30 yd. of the depth charge, and affected, dogs, goats, and monkeys.

4. Severe hæmorrhage was much more frequent in the right lung than in the left. In series 2, the right lung was more affected than the left in 62 per cent, the left more than the right in 8 per cent, both lungs equally affected in 30 per cent. In series 1, composed of goats only, the right lung was certainly more affected than the left in 50 per cent; in a further proportion we formed the impression that the right-sided lesions were somewhat more severe than those in the left lung.

The order of severity of lobar lesions was in series 1 (goats) right middle lobe, right lower, and right upper; in series 2 (goats, monkeys, and dogs) it was right upper and lower, right middle, postcaval, left upper, left middle and lower. Hilar hæmorrhage was found in 46 per cent of animals, being frequently continuous with more peripheral lesions.

The margins of the lungs were affected in all cases, the hæmorrhages being somewhat more common on the right side than the left. Such marginal hæmorrhages took the form of small wedges or linear areas, 1 to 3 cm. in greatest length, or of confluent petechiæ.

Similar small hæmorrhages and petechiæ were also common within the substance of the lung, especially at the margins of the large hæmorrhagic areas, but on the whole they were most numerous subpleurally.

5. Rib markings on the surface of the lungs were frequent in the goats of series 1, occurring in 50 per cent of animals. They were not nearly so obvious in the animals of series 2, but this may have been due to these animals being nearer the depth charge and suffering from more extensive hæmorrhage which masked the rib markings.

Table I.—MORTALITY RATES

	Total Number of Animals	Killed at Once	Survived	Percentage Dead
Above depth charge	1	1	0	100
20 yd. from depth charge	5	4	1	80
30 „ „ „ „	5	4	1	80
40 „ „ „ „	2	2	0	100
45 „ „ „ „	2	0	2	0
50 „ „ „ „	1	0	1	0
75 „ „ „ „	1	0	1	0
100 „ „ „ „	2	0	2	0
200 „ „ „ „	1	0	1	0
300 „ „ „ „	1	0	1	0
Total	21	11	10	52·4

Percentage dead at all distances 52·4
„ „ within 40 yd. 84·6

Table II.—INCIDENCE OF PULMONARY HÆMORRHAGE

	Total Number of Animals	Pulmonary Hæmorrhage			
		Severe	Moderate	Slight	Absent
Above depth charge	1	1	—	—	—
20 yd. from depth charge	5	2	2	1	—
30 „ „ „ „	5	2	2	1	—
40 „ „ „ „	2	—	1	1	—
45 „ „ „ „	2	—	1	1	—
50 „ „ „ „	1	—	1	—	—
75 „ „ „ „	1	—	—	1	—
100 „ „ „ „	2	—	—	2	—
200 „ „ „ „	1	—	—	—	1
300 „ „ „ „	1	—	—	—	1
Total	21	5	7	7	2

6. Petechiæ and small hæmorrhages were present in the walls of the trachea and bronchi in one goat of series 1 (20 yd.), and in 5 animals (1 dog at site of depth charge, 3 goats at 20 yd., 30 yd., and 30 yd. respectively (*Fig.* 61), and 1 monkey at 30 yd.), of series 2. The incidence for the whole series was thus 28·5 per cent.

7. Peculiar concentric lines of hæmorrhage were seen at the apices of 3 goats and 1 dog (20 to 30 yd. from depth charge), oval in shape, commencing near a large bronchus and extending towards the pleura (*Fig.* 62). These sometimes showed branches. The incidence of the rings was probably higher than these figures suggest, since a number of lungs were not completely dissected but saved as museum specimens.

FIG. 59.—Goat 2 (Series 1), 20 yd. from depth charge. Subpleural bullæ and interstitial emphysema with diffuse hæmorrhage.

FIG. 60.—Goat 4 (Series 2), 30 yd. from depth charge. Extensive pulmonary hæmorrhage, 'ring' formation in upper and middle lobes. Large bronchial clot.

FIG. 61.—Goat 3 (Series 2), 30 yd. from depth charge. Large diffuse hæmorrhage and petechiæ in tracheal wall.

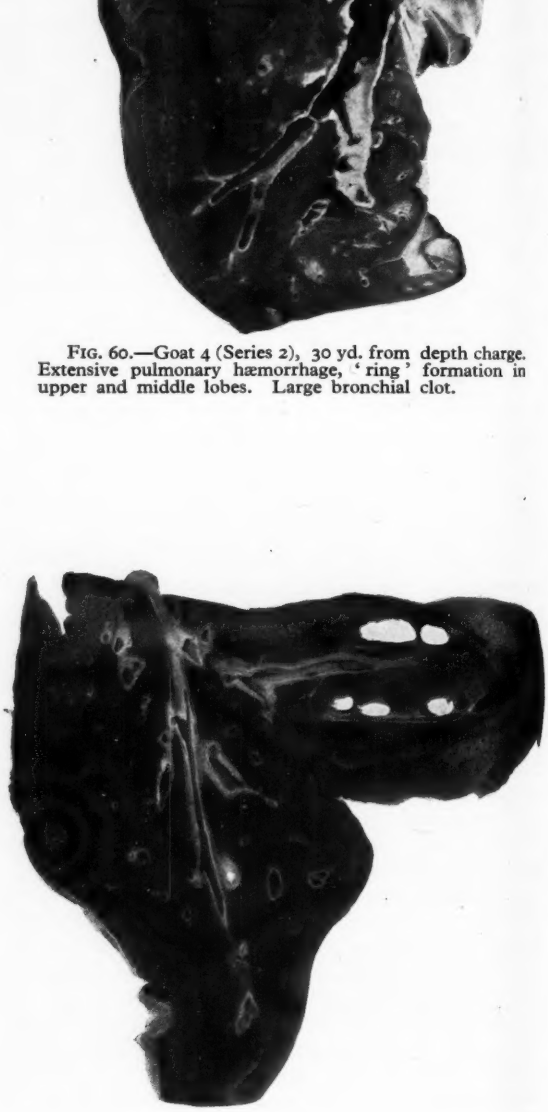

FIG. 62.—Dog 7, 20 yd. from depth charge. Concentric 'rings' in right middle lobe. Diffuse hæmorrhage in substance of lung.

8. Severe hæmorrhage with formation of large blood-clots was seen within the main bronchi and trachea in 6 animals, i.e., 28·5 per cent. This was invariably confined to animals within 30 yd. of the depth charge.

Acute emphysema was a striking feature macroscopically, resulting in the production of large air-containing vesicles within the substance of the lungs, numerous small vesicles along the interlobular septa and the bronchovascular planes of the lobes (acute interstitial emphysema), and of small and large subpleural bullæ.

1. Acute vesicular emphysema was present in all of the goats of series 1 (100 per cent) and in 61 per cent of animals of series 2. Acute interstitial emphysema was obvious in only one goat of series 1 (12·5 per cent), but was marked in 10 animals (77 per cent) in series 2. Sub-pleural bullæ were found in one goat (12·5 per cent) of series 1, in 8 animals of series 2 (61 per cent). *Tables III* and *IV* show the relation of incidence of emphysema and pneumothorax to distance from depth charge.

It appears that acute interstitial emphysema developed in animals within 40 yd. of the depth charge, whilst vesicular emphysema was seen in animals as far away as 100 yd. This agrees with the incidence of hæmorrhage in the lungs, severe and moderate hæmorrhage being seen up to 50 yd., slight hæmorrhage up to 100 yd.—in other words, within a radius of 40 to 50 yd. pulmonary injury is marked, resulting in extensive hæmorrhage in the lungs and rupture of air spaces with acute interstitial emphysema.

2. Emphysema was most frequent in the right and left upper lobes, least frequent in the lower and postcaval lobes. Acute interstitial emphysema was especially well marked in the interlobular septa and along the bronchi, sub-pleural bullæ on the ventral surface of the right lung towards the upper third.

3. No evidence of a species difference was obtained, but the number of animals in the groups was too small to draw conclusions.

4. Hæmothorax was uncommon, only two animals showing a small amount of blood in the pleural cavities.

Cardiovascular System.—Very little change was found in the cardiovascular system. Pericardial hæmorrhages were present in 8 (38 per cent), and were confined to animals within a radius of 40 yd. of the depth charge. Hæmorrhages were usually petechial, sometimes confluent, distributed along the coronary vessels towards the apex of the heart anteriorly and posteriorly. No leakage of blood into the pericardial sac had occurred. The heart was generally contracted. No other lesions were found. The large blood-vessels were unaffected throughout the body. No evidence of damage to mediastinal structures was seen.

Table III.—Incidence of Acute Emphysema and Pneumothorax

	Total Number of Animals	Acute Vesicular Emphysema Present	Acute Interstitial Emphysema Present	Pneumothorax Present
Above depth charge	1	1	1	1
20 yd. from depth charge	5	5	5	0
30 " " " "	5	5	4	2
40 " " " "	2	2	1	0
45 " " " "	2	2*	0	0
50 " " " "	1	1	0	0
75 " " " "	1	1	0	0
100 " " " "	2	2	0	0
200 " " " "	1	0	0	0
300 " " " "	1	0	0	0
Total	21	19	11	3

* Very slight, at margins of lungs.

Table IV.—Degree of Acute Interstitial Emphysema

(*Numbers in brackets indicate fatal cases*)

	Total Number of Animals	Acute Interstitial Emphysema			
		Severe	Moderate	Slight	Absent
Above depth charge	1	1 (1)	0	0	0
20 yd. from depth charge	5	1 (1)	1	3 (3)	0
30 " " " "	5	0	1 (1)	3 (3)	1
40 " " " "	2	0	1 (1)	0	1 (1)
45 " " " "	2	0	0	0	2
50 " " " "	1	0	0	0	1
75 " " " "	1	0	0	0	1
100 " " " "	2	0	0	0	2
200 " " " "	1	0	0	0	1
300 " " " "	1	0	0	0	1
Total	21	2	3	6	10

The Liver.—The liver was ruptured in the dog placed over the depth charge, its superior surface being lacerated in many places, both right and left lobes being involved. Blood had leaked into the peritoneal cavity, but the amount effused was not very great (about 1 oz.). Two goats showed small subcapsular ecchymoses. In all of the remaining animals the liver appeared unaffected.

The Pancreas.—Petechiæ were found in the pancreas of a monkey 40 yd. from the depth charge. The spleen showed small subcapsular hæmorrhages in 5 animals (24 per cent). It was usually small, sometimes pale and wrinkled, with little blood in its pulp.

The Kidneys.—The kidneys were normal in all animals with the exception of a small laceration at the left upper pole of the dog placed over the depth charge, and small capsular hæmorrhages in a monkey at 40 yd.

FIG. 63.—Dog 6, immediately above depth charge. Ruptured stomach.

The Alimentary Canal (*Table V*).—Lesions included the following :—

1. Rupture of the stomach in the dog placed over the depth charge (*Fig*. 63). This was due to a ragged longitudinal tear extending from the cardia to the pylorus on the anterior surface, the lumen gaping open and disclosing blood-stained gastric contents. A small perforation at the middle of the posterior surface was also present.

FIG. 64.—Monkey 13, 40 yd. from depth charge. Loops of small intestine showing numerous small mucosal hæmorrhages and ballooning of lumen.

2. Subserous hæmorrhages (up to 1 cm. diameter) in the stomach wall, towards the lesser and greater curvatures, in 3 animals (1 dog, 2 monkeys).

3. Hæmorrhage in the mucous coat of the small intestine of 3 monkeys (*Fig*. 64), in the mucosa of the large intestine of 2 monkeys, and in the rectum of 1 monkey. These hæmorrhages were generally multiple, varying from three to six, seldom exceeded 0·5 cm. diameter, tended to become confluent in the small intestine, and were normally associated with blood in the intestinal lumen. There was no evidence of perforation of the gut wall, but gross tearing of the mucosa had occurred in several instances. A striking feature was the distension of the gut lumen at the site of hæmorrhage, the dilated area of bowel contrasting with the narrowed portion above and below. The impression was formed that such areas might well have gone on to perforation if the animals had lived.

Table V.—INCIDENCE OF LESIONS OF THE ALIMENTARY CANAL

	STOMACH	SMALL INTESTINE	LARGE INTESTINE	NO LESIONS
Over depth charge	1	—	—	—
20 yd. from depth charge	1	—	—	4
30 " " " "	—	2	2	3
40 " " " "	1	1	2	0
45 " " " "	—	—	—	2
50 " " " "	—	—	—	1
75 " " " "	—	—	—	1
100 " " " "	—	—	—	2
200 " " " "	—	—	—	1
300 " " " "	—	—	—	1
Total	3	3	4	15

Table VI.—SPECIES INCIDENCE OF LESIONS OF THE ALIMENTARY CANAL

	Stomach Lesions	*Intestinal Lesions*
Monkey	1/4	4/4
Goat	0/12	0/12
Dog	2/3	0/3
Pig*	0/2	0/2

* Both animals were at a distance of 45 yd. from the depth charge, so that little can be attributed to this small group.

4. Hæmoperitoneum occurred in 1 animal only, the result of rupture of the liver.

5. A species difference seems to be present, since lesions of the alimentary canal were common in monkeys, sometimes seen in dogs, and were never seen in goats. *Table VI* summarizes these features. It must be emphasized, however, that with the exception of the animal over the depth charge, none of these lesions were extensive, although it was felt that some of them might have proved dangerous if the animals had survived.

An impressive feature was the manner in which *hollow viscera*, such as the gall-bladder, urinary bladder, and renal pelves, escaped injury, even when these were partly distended with bile or urine. This is in striking contrast with the behaviour of air-containing viscera, which often showed damage.

Ductless Glands rarely showed lesions. Hæmorrhage and pulping of the adrenals were seen in the dog placed over the depth charge and petechiæ were found in the thymus of a monkey at 40 yd. from the depth charge. No other ductless gland was affected, neither were the reproductive organs injured.

The Blood.—No characteristic change was noted in the blood, which, however, was invariably fluid at the post-mortem examination two to four hours after death.

The central nervous system was not examined by us.

MORBID HISTOLOGY

Histological examination has confirmed the impression received by naked-eye examination that the most important structural changes produced by a depth charge are found in the respiratory system. If has, moreover, become certain

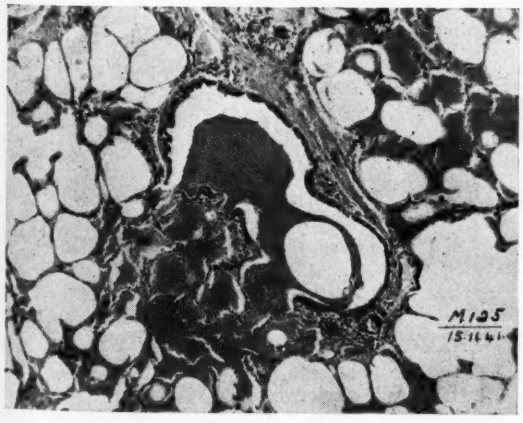

FIG. 65.—Goat 7 (Series 1), 100 yd. from depth charge. Lobular hæmorrhage, with blood entering a bronchiole. (Ehrlich's acid hæmatoxylin and eosin.) (× 45.)

that there is a striking similarity in the nature of these effects, the main variation being in their intensity and distribution. Microscopical details of individual cases are therefore not included in this report, since there is little to add to the anatomical description so far as variation from case to case is concerned.

Pulmonary Changes fall into two main groups: (1) Hæmorrhage; and (2) Emphysema with rupture of air spaces. It is our intention to discuss these in some detail and to show how

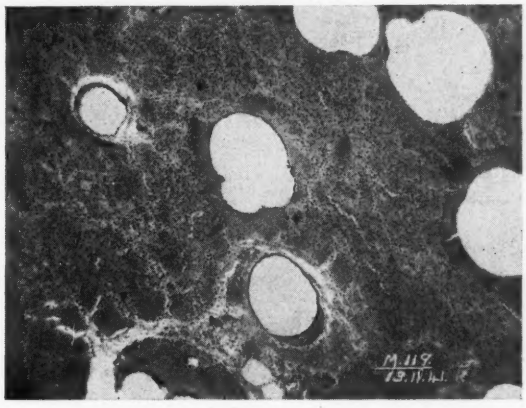

FIG. 66.—Goat 2 (Series 1), 20 yd. from depth charge. Severe pulmonary hæmorrhage. (Ehrlich's acid hæmatoxylin and eosin.) (× 45.)

far their study leads to an elucidation of the mechanism of depth-charge effects.

Hæmorrhage.—It becomes apparent at the commencement of histological examination, that hæmorrhage falls into two groups: (1) small to medium-sized, and (2) massive.

Small and medium hæmorrhages are essentially similar in nature, differing only in their extent. There is no distinction in their distribution in the lung lobule. The smallest hæmorrhages occupy one or several alveoli, being made up of clumps or closely packed masses of red blood-corpuscles with well-preserved outlines, no shrinkage or distortion, and staining normally (*Fig.* 65). There is little associated coagulum or fibrin. Capillaries in the alveolar walls are distended with corpuscles, and it is not difficult to trace the effused erythrocytes to the capillaries. There is little difficulty, too, in finding tears and complete ruptures of the alveolar walls. Medium-sized hæmorrhages involve many more alveoli and may include infundibular ducts and even terminal bronchioles. The typical picture is one of a number of alveoli filled with corpuscles grouped irregularly or in continuity around infundibula or bronchioles, the lumina of which may be loosely or tightly packed with red clot. Rupture of the infundibular or much less frequently the bronchiolar wall can occasionally be made out, and no doubt has been concerned in the production of hæmorrhage. It must be emphasized that a particular hæmorrhage is always confined to a pulmonary unit, the lobule.

Massive hæmorrhage of the type which has been likened to an infarct is just as definite in its anatomy (*Fig.* 66). We have little doubt that it represents the coalition of a large number

of small hæmorrhages, and as such has essentially the same sort of origin as these. It is doubtful how far the analogy to a true pulmonary infarct, of arterial origin, can be pushed. It is true that the essential features of infarction are present. Large tracts of lung tissue are tightly packed with red corpuscles, and the outlines of the air spaces, though obscured in this way, can still be made out. On the other hand, no apex and base is apparent. We are well aware that too much prominence should not be placed on these latter features, but in so far as they indicate the essential characteristic of a true infarct, namely, a topography determined by arterial distribution, we feel justified in stressing their importance in our analysis. Depth-charge 'infarcts', on the other

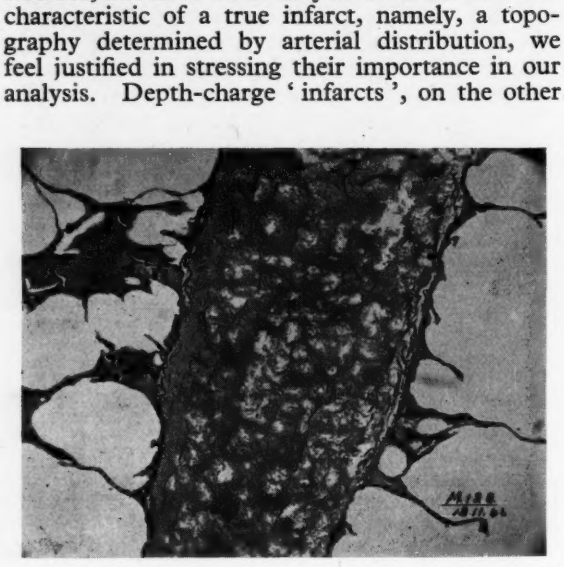

FIG. 67.—Goat 3 (Series 2), 30 yd. from depth charge. Exudate containing protein, but no red blood-cells, in interstitial plane. Ruptured lymphatics. (Ehrlich's acid hæmatoxylin and eosin.) (× 45.)

hand, seldom show such a clear apex and base. When they do, the apex turns out to be a bronchus. Here, no less than in the small hæmorrhages, the distribution of blood is essentially lobular. A study of serial sections has convinced us that hæmorrhage arises within the air spaces, in most cases spreading from the periphery of lobules centripetally to the terminal and larger bronchi. The supporting tissue around the arteries and arterioles is seldom the site of red-cell effusion. Although perivascular spaces are sometimes filled with blood, this is the exception rather than the rule. Prolonged search, moreover, has failed to disclose any break in the walls of arteries or veins, although we have noted fairly often red cells in the walls of these structures. Massive hæmorrhage, therefore, is produced through the involvement of a large number of lung lobules. These generally follow the distribution of a bronchus, but not infrequently a series of lobules impingeing on an interlobular septum or the pleura is involved in hæmorrhage.

Septal hæmorrhage is no less interesting. In the most severe cases, septa may be completely infiltrated with blood, which can be traced for long distances and may eventually reach the pleura. The origin of the hæmorrhage is often enough obscure. It is possible at times to be certain that capillaries and small veins or arteries of the septa have ruptured, but it is equally certain that sometimes blood from neighbouring injured alveolar walls has broken through into the septa. A similar state of affairs seems to hold for hæmorrhage in the perivascular and peribronchial spaces. We would call attention, however, to the frequency with which effusion of lymph from the strikingly distended lymphatics occurs (*Fig.* 67). In the absence of lymphatic obstruction, such as might occur from the marked accumulation of air as the result of acute interstitial emphysema, a state of affairs we have never seen, such lymphatic rupture must surely be due to direct trauma, the result of the depth charge. Since lymphatic injury is demonstrable, we think that capillary injury is no less likely to occur.

A further point of interest is the extensive congestion which is widely diffused throughout the lungs. Except in areas of vesicular emphysema where capillaries are stretched and often appear almost empty of blood, all types of small vessels are dilated and commonly filled with corpuscles and plasma. Large vessels, on the other hand, are frequently empty, but little importance should be attached to this fact since no precaution as to tying off vessels at the autopsy was taken.

Hæmorrhage in the walls of the bronchi or the trachea was seen in a proportion of cases. This affected the mucous membrane mostly, being subepithelial and occasionally bursting through into the lumen. In some instances an association with the cartilage rings could be made out, whilst small effusions of red cells within bronchial muscle and mucous glands were seen. The hæmorrhages thus appeared to be definite entities independent of the changes in neighbouring lobules. They probably are to be attributed to the direct action of the depth charge, although disturbance of bronchial circulation secondary to the severe pulmonary circulation upset might be responsible.

It is pertinent to inquire at this stage into the cause of pulmonary hæmorrhage following depth-charge exposure. Histological investigation suggests that it is mainly capillary, since hæmorrhage is essentially most severe in those regions where the capillaries are most numerous. There appears to be a wholesale escape of blood-cells to a degree difficult to understand under ordinary conditions of circulation. For this reason, attention must be directed to the pulmonary congestion, a feature which might well be invoked to explain the considerable loss of blood from injured capillaries. Rupture of a certain number of capillaries can be made out, but taken as a whole seems insufficient to account for the extent of blood-loss. We are forced to fall back on the idea that many of the corpuscles have been forced

through the walls of congested capillaries (diapedesis) as occurs from time to time in inflammatory conditions of the lungs. Such a view seems the more acceptable in the light of modern ideas of capillary behaviour after puncture or tearing, since there is considerable evidence that in such circumstances hæmostasis is maintained by retraction and obliteration of the damaged vessel (*see* McFarlane, *Quart. Jour. Med.*, 1941, N.S., **10**, 1, for review).

Emphysema.—This type of lesion falls quite clearly into two groups: (1) acute vesicular emphysema, and (2) acute interstitial emphysema with tracking of air along the interstitial tissues and pleura of the lung.

Vesicular emphysema affects groups of alveoli, infundibula with their alveoli, or may be associated

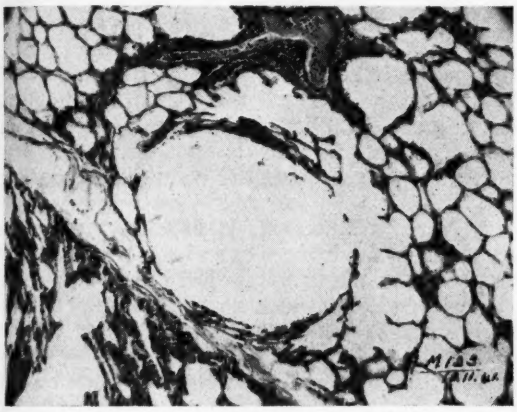

FIG. 68.—Goat 4 (Series 1), 50 yd. from depth charge. Vesicular emphysema involving infundibula and alveolar ducts. (Ehrlich's acid hæmatoxylin and eosin.) (× 45.)

with some bronchiolar dilatation. Generally speaking, infundibula and alveoli are the structures most affected (*Fig.* 68). Such changes are seen in the areas of hæmorrhage, where the emphysematous cavities may stand out as empty spaces or may be partly filled with blood. A certain amount of distension occurs, too, around hæmorrhagic areas and even at some distance from these. Tearing of alveolar walls may be seen, with intercommunication between neighbouring alveoli so that large air sacs develop. Some of these almost certainly arise through rupture of infundibular ducts or stretching of terminal bronchioles (*Fig.* 69).

Interstitial emphysema is a striking feature in animals within 30 or 40 yd. of the depth charge. It occurs in the form of a definite pattern, small air cavities being seen most often in the interlobular and interlobar septa and within the supporting tissue around the main bronchi and blood-vessels. The pleura becomes involved and large subpleural bullæ form (*Fig.* 70).

In the most severe cases large cavities form close to the larger bronchi. These show a good deal of disruption of the lung, whole lobules bursting and losing their constituent structures. It is possible to follow the mode of development of interstitial emphysema, which seems to commence through bursting of alveoli adjacent to

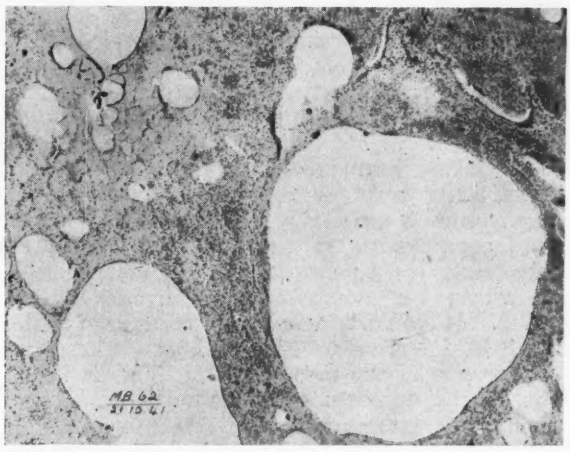

FIG. 69.—Goat 2 (Series 1), 20 yd. from depth charge. Gross vesicular emphysema of infundibular duct, with surrounding hæmorrhage. (Ehrlich's acid hæmatoxylin and eosin.) (× 60.)

septa or pleuræ. Ragged remains of alveolar or infundibular duct walls are seen. Small gaps can be made out quite easily between main infundibula or alveoli and septa and can be traced in continuity with septal or subpleural

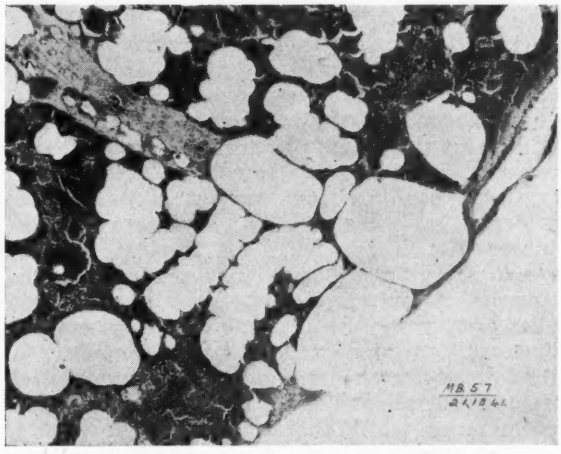

FIG. 70.—Goat 2 (Series 1), 20 yd. from depth charge. Subpleural emphysematous bullæ and interstitial emphysema associated with tears of the alveolar walls. (Ehrlich's acid hæmatoxylin and eosin.) (× 50.)

air bubbles. A certain amount of tearing of interstitial tissue occurs and may lead, in the very severe cases, to considerable disruption of septal or pleural tissue. Tracking of air alongside the larger blood-vessels and bronchi has been seen, but only to a slight degree. This, no doubt, was restricted by the short duration of the experiment, no animals surviving longer than

about 11 min. after the explosion of the depth charge. For this reason, nothing resembling an air lock at the hilum (Macklin, *Trans. Roy. Soc. Canad.*, 3rd sev., **34**, s.v, 69) was encountered.

Relationship between Hæmorrhage and Emphysema.—A close study of serial sections from a number of affected areas has convinced us that hæmorrhage can occur quite independently of emphysema. In many areas numerous small and confluent hæmorrhages occupy large numbers of alveoli and extend throughout many lobules without the slightest sign of emphysema. In the monkey, hæmorrhage may be very severe, but vesicular emphysema is slight and interstitial emphysema is practically absent. On the other hand, emphysema is nearly always associated with extensive hæmorrhage in adjacent alveoli or within the emphysematous cavities themselves. It would appear that hæmorrhage is the result of a less severe type of injury, interstitial emphysema of the most serious. This is borne out by the occurrence of hæmorrhage and the absence of interstitial emphysema in lungs of animals at some distance from the depth charge. Vesicular emphysema, on the other hand, is common at all distances, being invariably associated with hæmorrhage. It represents either a less severe form of injury of the air spaces or a compensatory reaction to loss of lung tissues from hæmorrhage.

Origin of Bronchial and Tracheal Clots.—These are the result of hæmorrhage in the lobules, since they can in most cases be traced from the periphery of lobules to the main bronchi. Tears with a resulting hæmorrhage in infundibular ducts and bronchial walls also contribute to the formation of such clots. Aspiration of blood into adjacent lobules no doubt plays a part as well.

Changes in Other Organs.—Little need be said about the histology of the changes in other organs, since in most cases these consist of capillary hæmorrhages without any clear-cut evidence of tearing of such vessels. Small subcapsular hæmorrhages in the spleen arise within the pulp through injury of the peripheral sinuses. A certain amount of congestion has been seen in the medulla of the kidneys and the liver. In the one instance of severe injury of the liver, numerous large areas of tearing occurred near the surface and around the hepatic veins and portal canals within the substance of the organ.

The gastric and intestinal hæmorrhages commence as small mucosal and adventitial petechiæ or diffuse mucosal infiltrations with tearing of the surface epithelium through which the blood-cells pour into the gut lumen.

No evidence of fat embolism was found in the organs of animals exposed to depth charge.

SUMMARY

1. Animals exposed to a depth charge develop severe pulmonary hæmorrhage and interstitial emphysema, sometimes hæmorrhage and tearing in the wall of the alimentary canal, occasionally hæmorrhage in the epicardium, spleen, kidneys, and ductless glands.

2. The most severe injury is seen in animals within a radius of 40 yd. from the depth charge. Eleven of 13 animals within this radius were killed instantaneously. Ten showed extensive pulmonary hæmorrhage, 5 moderate to severe interstitial emphysema. Hæmorrhage may, however, develop in animals as far away as 100 yd.

3. Severe pulmonary hæmorrhage may occur without immediate death, whilst animals may be killed at once and not show marked pulmonary lesions.

Hæmorrhage affects more frequently the right lung than the left, the upper lobes and margins of the lung being the most common sites involved. Bronchi may become filled with large blood-clots, whilst small hæmorrhages sometimes develop in the walls of the trachea and bronchi.

5. Acute vesicular and interstitial emphysema are practically constant in animals within 40 yd. of the depth charge. Emphysema affects the upper lobes of both lungs most often, being least frequent in the lower lobes. Pneumothorax and hæmothorax are sometimes associated with it.

6. Very little damage is produced in the soft structures of the body wall and the bones. There is no special tendency for certain hollow fluid-containing viscera, such as the gall-bladder and urinary bladder, to be involved.

We wish to thank the many people who have helped us in this investigation.

First, Surgeon Captain Fairley, R.N., Superintendent of the Physiological Section, C.D. Experimental Station, Porton.

Also Captain Blount, R.A.V.C., for assistance.

We should also like to thank Messrs. Burgess and Allan for their support in the post-mortem room.

Also Mr. R. McV. Weston, Superintendent of the Photographic Department, Porton, for the trouble he has taken with the photographs.

APPENDIX

Series 1

20 yd. from Depth Charge.—

Goat 2.—In sea from 1.27 p.m. to 1.48 p.m. Dead on removal. Post-mortem examination about 1½ hr. later. No external signs of injury.

Heart: Two ventral hæmorrhages. The first (about 1 cm. diameter) on left ventricle near the left coronary artery; the other (about 2 cm. diameter) on the wall of the right ventricle. Several petechial hæmorrhages scattered over the surface near the base of the heart. No petechiæ in parietal pericardial sac or in endocardium.

Lungs: Nearly ½ pint of blood-stained fluid in each pleural cavity. Lungs solid, with massive hæmorrhages involving about three-quarters of their total volume. The apices and bases less massively involved. Bases paler, but solid to the feel. Rib markings along dorsal surface of both lungs. Many

subpleural and interstitial emphysematous bullæ in scattered groups over the surface of both lungs, the largest containing about 3 c.c. of air and the smallest about 0·5 cm. diameter (*see Fig.* 59). More numerous on the ventral than the dorsal surface. Cut surface of lungs showed the hæmorrhage to be even more extensive than had been suspected, the apices and bases being hæmorrhagic. Hæmorrhage was intensest, consisting of frank blood with no macroscopic indication of lung tissue, in zones following approximately the bronchial tree, into which it had burst and filled the lumen, with clot in the bronchi and blood-stained frothy fluid in the trachea. Large emphysematous bullæ scattered through lung substance, often near a bronchus. Impossible to demonstrate connexion with bronchus. Elsewhere saccular dilatations of bronchi.

Trachea and Bronchi: Blood-stained frothy fluid in trachea. Clots in all larger bronchi. A few small petechial hæmorrhages in bronchial wall beneath mucous membrane, and extending upwards to lower third of trachea.

Chest Wall and Diaphragm: No signs of injury or bruising.

Liver: One small ecchymosis on left posterior free border.

Pancreas: Nil.
Spleen: Nil.
Kidneys: Nil.
Alimentary Canal: Nil.
Bladder: Full, normal.

30 yd. from Depth Charge.—

Goat 3.—In sea from 1.26 p.m. to 1.50 p.m. Alive on removal. Shot by humane killer. Post-mortem examination about 1½ hr. later. No external signs of injury.

Heart: Small ecchymoses and petechial hæmorrhages around base of aorta.

Lungs: Massive hæmorrhages involving whole of both middle lobes and adjacent parts of upper and lower lobes. Blood-clot in whole of bronchial tree to middle lobe and in branch to upper segment of lower lobe. Elsewhere lung substance congested, with scattered discrete hæmorrhages, sometimes coalescing to form larger areas of hæmorrhage in upper lobe, but to a less extent in lower lobe. No bullous emphysema. Vesicular emphysema, particularly at free margins.

Trachea and Bronchi: No hæmorrhages in mucous membrane of trachea. Blood-clot in small bronchi.

Chest Wall and Diaphragm: No signs of bruising or injury.

Liver: Pedunculated cyst (cysticercus) hanging from anterior inferior surface of liver. Occupying area where this cyst was in contact with the liver there was a bruise, size of a sixpence, subcapsular and extending a short way (under 0·5 mm.) into liver substance.

Pancreas: Nil.
Spleen: Nil.
Kidneys: Nil.
Bladder: Full, no injury.
Alimentary Canal: Nil.

50 yd. from Depth Charge.—

Goat 4.—In water from 1.24 p.m. to 1.51 p.m. Alive on removal. Shot with humane killer. Post-mortem examination about 1½ hr. later. No external signs of injury.

Heart: No petechiæ.

Lungs: Rib markings on convex surface and on the ventral surface of the middle lobes. No hæmorrhages on surfaces or cut surfaces of either lung. Congestion following bronchial tree. Bases firm to feel, ? collapse, with parasitic nodule at base of right lower lobe.

Trachea and Bronchi: No fluid. No hæmorrhages.

Diaphragm, Abdominal Wall, and Chest Wall: Nil.
Liver: Nil.
Pancreas: Nil.
Spleen: Nil.
Kidneys: Nil.
Bladder: Full, normal.
Alimentary Canal: Nil.

75 yd. from Depth Charge.—

Goat 6.—In water from 1.22 p.m. to 1.52 p.m. Alive on removal Shot. Post-mortem examination about 2 hr. later.

No external signs of injury.

Heart: Scattered subepicardial petechiæ on surface. Nil in endocardium.

Lungs: Scattered discrete hæmorrhages over subpleural surfaces of all lobes. Rib markings on dorsal surface. Livery consistence at both bases. Green discoloration of lower lobe due to inhaled stomach contents.

Trachea and Bronchi: Froth. Inhaled sea water —diatoms present. No hæmorrhages.

Chest Wall, Abdominal Wall, and Diaphragm: Nil.
Liver: Nil.
Spleen: Nil.
Pancreas: Nil.
Kidneys: Nil.
Bladder: Nil.
Alimentary Canal: Nil.

100 yd. from Depth Charge.—

Goats 7 *and* 10.—Goat 7 in water from 1.19 p.m. to 1.56 p.m. Goat 10 from 1.19 p.m. to 1.56½ p.m. Both alive on removal. Both shot. Post-mortem examination about 2½ hr. later.

External Injuries: Goat 7, fractured pelvis; Goat 10, nil.

Heart: Petechiæ at base around aorta and pulmonary artery of both.

Lungs: Small hæmorrhages, subpleural or interstitial, about 1 cm. diameter scattered over the surfaces of both lungs, either singly or in groups. Hyperæmia at base of lower lobe of right lung of Goat 7. Marginal emphysema. Livery consistence at bases.

Trachea and Bronchi: Inhaled stomach contents in trachea of goat 7 and evidence of sea-water diatoms present.

Diaphragm, Abdominal Wall, and Chest Wall: Nil.
Liver: Nil.
Pancreas: Nil.
Spleen: Nil.
Kidneys: Nil.
Bladder: Nil.
Alimentary Canal: Nil.

200 yd. from Depth Charge.—

Goat 11.—In water from 1.15 p.m. to 1.57 p.m. Alive on removal, shot. Post-mortem examination about 2½ hr. later.

Heart: Nil.

Lungs: Two small hæmorrhages at lower border of upper right lobe. Scattered areas of congestion throughout cut surface. Marginal vesicular emphysema. Livery feel at bases.

Trachea and Bronchi: Nil. Sea water inhaled—diatoms present.
Abdominal Wall, Chest Wall, and Diaphragm: Nil.
Liver: Nil.
Pancreas: Nil.
Spleen: Nil.
Bladder: Nil.
Kidneys: Nil.

300 yd. from Depth Charge (supposed control).—

Goat 12.—In water from 1.37 p.m. to 1.40 p.m. Alive on removal, shot. Post-mortem examination about 2 hr. later. No external signs of injury.
Heart: Nil.
Lungs: Four groups of scattered small hæmorrhages in upper (1), middle (2), and lower (1) lobes. Livery feel at both bases. Marginal vesicular emphysema.
Trachea and Bronchi: Nil—no fluid present.
Liver: Nil.
Pancreas: Nil.
Spleen: Nil.
Kidneys: Nil.
Bladder: Nil.

SERIES 2

Immediately above Depth Charge.—

Dog 6.—No protection. In sea from 12.47 p.m. to 1.10 p.m. approximately. Found dead. Post-mortem examination about 3 hr. later. No signs of external injury. No hæmorrhages in skin, subcutaneous tissues, or muscles of thorax and abdomen. No bleeding from orifices. Eyes normal.
Heart: Large petechiæ and hæmorrhages in pericardium, especially along coronary vessels and on right auricle. A few on left auricle. Very little pericardial fluid, clear. Myocardium normal. Cavities slightly dilated or in diastole. No abnormalities of valves or papillary muscles. No displacement of heart. No air in cavities. Large blood-vessels normal.
Lungs: About 1 oz. of blood in both pleural cavities, apparently coming from torn lung. Old pleural adhesions attaching right posterior lung to chest wall. Pneumothorax on both sides. Extensive hæmorrhage, tearing, and emphysema affecting whole of both lungs, more severe on right side. Lung substance almost black, with blood-like massive infarct; friable, tearing easily when removed. Huge cavities filled with air and blood, extending to pleura, with large subpleural bullæ. Blood in main bronchi.
Trachea: Contained blood-stained frothy fluid. Petechiæ in wall above bifurcation.
Liver: Extensive tearing with rupture of convex surface (subdiaphragmatic mostly), extending across both lobes, more marked on right side. Anterior margin unaffected. Inferior surface showed a few small lacerations. A little clotted blood attached to torn areas, suggesting that death had been instantaneous. Gall-bladder moderately distended with green bile. Wall normal.
Pancreas: Normal.
Spleen: Congested, but not very large. No evidence of rupture, but some small subcapsular hæmorrhages of recent origin along anterior surface.
Kidneys: At upper pole of left kidney on anterior surface a recent tear about 1 cm. long, extending into substance of cortex. Small blood-clot attached. Remainder of kidney appeared normal. Right kidney normal.
Bladder: Empty; wall normal.
Genital Organs: Normal.
Ductless Glands: Thymus normal, atrophied. Left adrenal pulped and hæmorrhagic, so that recognition of any structure difficult. Right adrenal normal. Thyroid normal.
Alimentary Canal: A little recent blood-clot in preitoneal cavity (not more than 1 oz.) attached to clot on liver. Stomach showed large ragged tear along greater part of anterior surface, so that lumen gaped open, disclosing blood-stained gastric contents (*see Fig.* 63). Small perforation on posterior surface about middle. No abnormality in small and large intestines.
Bones: No fracture in long bones and ribs. Pelvis appeared unaffected so far as could be detected.
Blood: Still unclotted in most vessels at autopsy.
Central Nervous System: Not examined.
SUMMARY OF PATHOLOGICAL FINDINGS.—Massive hæmorrhage and laceration, with acute emphysema of both lungs; hæmo- and pneumothorax; laceration and rupture of liver, left kidney, and left adrenal; ruptured stomach; hæmoperitoneum; subcapsular hæmorrhages in spleen; pericardial hæmorrhages.

20 yd. from Depth Charge.—

Dog 7.—Abdomen protected. In sea from 12.49 p.m. to 1.13 p.m. approximately. Killed 13 min. after explosion. Post-mortem examination about 3 hr. later. No signs of external injury. No hæmorrhage in skin, subcutaneous tissues, or muscles of thorax and abdomen. No bleeding from orifices. Eyes normal.
Heart: No petechiæ in pericardium. Myocardium normal, in systole. Valves and papillary muscles normal. Pericardial fluid not excessive, clear. No displacement. No air in cavities. Large blood-vessels normal.
Lungs: Pleural cavities free from blood and air. No subpleural tearing or hæmorrhage. Diffuse congestion of both lungs with a dark-red hue. Large areas of hæmorrhage around margins, extending about 1 to 3 cm. into depth of lungs, most marked in middle and lower lobes. A few emphysematous bullæ at edges, with tiny blebs along interlobular fissures and scattered throughout middle and upper lobes. No rib markings discernible. Blood in smaller bronchi. Concentric hæmorrhagic rings at apices of right upper and middle lobes (*see Fig.* 64).
Trachea: Empty. No hæmorrhages in wall.
Liver: Normal.
Gall-bladder: Normal.
Pancreas: Normal.
Kidneys: Normal.
Spleen: Two small hæmorrhages (0·5 cm. diam.) close to hilar vessels. Spleen otherwise normal.
Bladder: Contained a little clear urine. Wall normal.
Genital Organs: Normal.
Ductless Glands: Normal.
Alimentary Canal: No free blood in peritoneal cavity. Stomach showed two oval-shaped hæmorrhages (1 to 2 cm. diam.) on posterior surface, one near middle of lesser curvature, the other about the middle of the posterior surface, subserous. Small and large intestines normal.
Bones: No fracture in long bones, ribs, or pelvis.
Blood: Still fluid in some of the larger vessels of the thorax and abdomen.
Urine: Clear. No abnormality in fæces.
Central Nervous System: Not examined.
SUMMARY OF PATHOLOGICAL FINDINGS.—Severe hæmorrhage and acute emphysema of lungs; subpleural interstitial emphysema; small subcapsular hæmorrhages in spleen; subserous hæmorrhages in stomach.

Dog 8.—Chest protected. In sea from 12.49 p.m. to 1.13 p.m. approximately. Found dead. Post-mortem examination about 3½ hr. later. Blood oozing from both nostrils, but not from other orifices. No hæmorrhage in skin, subcutaneous tissue, or muscle. Eyes normal.

Heart: Multiple small and large hæmorrhages in visceral pericardium. Pericardial fluid clear, not excessive. Endocardium and myocardium normal. Heart in systole. Valves and papiliary muscles normal. No air in cavities. No displacement of heart. Large blood-vessels normal.

Lungs: Pleural cavity contained no free blood. Parietal layer not torn or bruised. Numerous small hæmorrhages on visceral layer. No air in pleural cavity. Whole of both lungs occupied by massive black hæmorrhage, barely any part unaffected. Hæmorrhage least marked towards hila. Some blood in smaller bronchi, but not in main bronchi. No gross emphysema; numerous small vesicles along interlobular fissures of right and left upper lobes.

Trachea: Contained frothy fluid only. No hæmorrhages in wall.

Liver: Normal.
Gall-bladder: Normal.
Pancreas: Normal.
Spleen: Normal.
Kidneys: Normal.
Genital Organs: Normal.
Ductless Glands: Normal.

Bladder: Moderately distended with clear urine. Wall normal.

Alimentary Canal: Normal throughout. No free blood in peritoneal cavity.

Bones: No fractures in long bones, ribs, or pelvis.
Blood: Clotted throughout vessels.
Central Nervous System: Not examined.

SUMMARY OF PATHOLOGICAL FINDINGS.—Massive hæmorrhage in both lungs; slight interstitial emphysema; multiple subpericardial hæmorrhages; epistaxis.

Goat 5.—No protection. In sea from 12.33 p.m. to 1.13 p.m. approximately. Found dead. Post-mortem examination about 2 hr. later. No signs of external injury. No bleeding from orifices. No hæmorrhages in skin, subcutaneous tissue, or muscle. Eyes normal.

Heart: No petechiæ or hæmorrhages in pericardium or endocardium. Myocardium normal. Heart in systole. Valves and papillary muscles normal. Coronaries normal. No air in cavities. No displacement of heart. Pericardial fluid very scanty, clear. Large blood-vessels normal.

Lungs: Pleural cavities empty, no air. No tearing of parietal pleura. Relatively little hæmorrhage in lungs, chiefly in upper and middle lobes near hilum, with a few small scattered subpleural hæmorrhages near margins. One large hæmorrhage (about 2 cm. × 1 cm.) near notch of left upper lobe just below surface. No trace of rib markings. No obvious emphysema, but a few tiny bubbles along the interlobular septa of upper right lobe on fixing. No blood-clot in bronchi.

Trachea: Contained a little frothy fluid, not blood-stained. No hæmorrhages in walls.

Liver: Normal.
Gall-bladder: Normal.
Pancreas: Normal.
Kidneys: Normal.
Genital Organs: Normal.
Ductless Glands: Normal.

Spleen: Flattened, with a wrinkled pale surface; contained little blood on section.

Bladder: Partly distended with clear urine, wall normal.

Alimentary Canal: No free blood in peritoneal cavity. No lesions anywhere.

Bones: No fractures in long bones, ribs, or pelvis.
Blood: Still fluid.
Central Nervous System: Not examined.

SUMMARY OF PATHOLOGICAL FINDINGS.—Slight hæmorrhage in both lungs; slight interstitial emphysema.

Goat 6.—No protection. In sea from 12.30 p.m. to 1.13 p.m. approximately. *In extremis* 11 min. afterwards. Post-mortem examination about 2¼ hr. later. No signs of external injury. No bleeding from orifices. No hæmorhage in skin, subcutaneous tissue, or muscle.

Heart: No petechiæ in pericardium or endocardium. Muscle normal. Valves and papillary muscles normal. No air in cavities. No displacement of heart. Large blood-vessels normal. Heart in systole.

Lungs: Clear pleural fluid, yellow, in right chest cavity, about 2 to 3 oz. No air in pleural cavities. Extensive black hæmorrhage throughout greater portion of all lobes of right lung, with large clots in main bronchi. Some emphysema at edges of right middle lobe. One large hæmorrhage subpleurally at left apex and in middle lobe. Large blood-clots in main bronchi of all lobes, extending from clot in trachea. Since the left lower lobe is uninjured it follows that these bronchial clots are the result of aspiration. Huge clot in right and left main bronchi extending upwards for some distance into trachea. Numerous petechiæ in wall of main bronchi and lower trachea, submucous in position. A line of petechiæ extending along midline of anterior surface of trachea for about 4 cm. at level of middle of trachea. Concentric hæmorrhagic rings at both apices, oval shaped.

Liver: Normal.
Gall-bladder: Normal.
Pancreas: Normal.
Kidneys: Normal.
Genital Organs: Normal.
Ductless Glands: Normal.

Spleen: Flattened, pale, almost bloodless. No hæmorrhages.

Bladder: Moderately distended with clear urine. Wall normal.

Alimentary Canal: No lesions anywhere. No free blood in peritoneal cavity. Lymph-glands of mesentery large, with much lymph in sinuses.

Bones: No fracture in long bones, ribs, or pelvis.
Blood: Fluid.
Central Nervous System: Not examined.

SUMMARY OF PATHOLOGICAL FINDINGS.—Extensive hæmorrhage of both lungs; hæmorrhage into bronchi and trachea; acute interstitial emphysema; petechiæ in walls of bronchi and trachea.

30 yd. from Depth Charge.—

Goat 3.—Abdomen protected. In sea from 12.30 p.m. to 1.15 p.m. approximately. Found dead. Post-mortem examination 2¼ hr. later. Hæmorrhage from both nostrils. No other evidence of external injury. Skin, subcutaneous tissue, and muscles showed no hæmorrhage. Eyes normal.

Heart: Small hæmorrhage (0·5 cm. × 0·2 cm.) in visceral pericardium near apex of left ventricle

anteriorly. Endocardium and myocardium, valves, and papillary muscles normal. No displacement of heart. No air in cavities. Heart in systole. Large blood-vessels normal.

Lungs: Pleural cavities free from blood, parietal pleura showed no hæmorrhage. Air in both cavities. Large diffuse hæmorrhage and petechiæ in tracheal wall between cartilage rings, extending from 16th to 31st rings (*see Fig.* 61). These were towards lateral margins and on both anterior and posterior surfaces. Some slight bleeding had occurred into lumen from these. Numerous petechiæ in walls of main bronchi of both lungs. Extensive hæmorrhage throughout all lobes of right lung and in upper and middle left lobes, with moderate hæmorrhage in left lower lobe. Curious concentric markings, branching and converging to the apex, in upper and middle lobes of both lungs, due to maximal hæmorrhage. Broad rib markings at base of right lower lobe, imperfectly formed. Emphysema marked in left upper lobe, but very slight in right upper and middle lobes. No large blood-clot in bronchi.

Liver: Normal.
Gall-bladder: Normal.
Pancreas: Normal.
Spleen: Normal.
Kidneys: Normal.
Genital Organs: Normal.
Ductless Glands: Normal.
Bladder: Contained a little clear urine, wall normal.
Alimentary Canal: No lesions in stomach or intestines. No blood in peritoneal cavity.
Bones: No fractures in long bones, ribs, or pelvis.
Blood: Fluid.
Central Nervous System: Not examined.

SUMMARY OF PATHOLOGICAL FINDINGS.—Extensive hæmorrhage in both lungs; tracheal and bronchial petechiæ and submucous hæmorrhages; pneumothorax; slight acute emphysema; pericardial petechiæ; epistaxis.

Goat 4.—Chest protected. In sea from 12.30 p.m. to 1.15 p.m. approximately. Found dead. Post-mortem examination about 2½ hr. later. No evidence of external injury. No hæmorrhage in skin, subcutaneous tissues, or muscles. Eyes normal. No bleeding from orifices.

Heart: Two small hæmorrhages on pericardium near apex of left ventricle. Pericardial fluid scanty, no free blood. No subendocardial hæmorrhage. Myocardium, valves, papillary muscles normal. No air in cavities. Heart not displaced, in systole. Large blood-vessels normal.

Lungs: Pleural cavities contained no blood, but air in left cavity. No tearing or hæmorrhage in pleuræ. Massive blood-clots in both main bronchi extending into trachea. Patches of petechiæ in wall of trachea between 25th and 28th cartilages and around 35th cartilage. No petechiæ in main bronchi. Massive hæmorrhage in right lung, especially throughout upper and middle lobes and along anterior margin of lower lobe. A few large hæmorrhages along margins of left middle and lower lobes, not extending more than 1 to 2 cm. into substance from pleura. Concentric hæmorrhagic rings at apices of upper and middle lobes. Large areas of emphysema in upper and middle lobes of right and left lungs, several large subpleural bullæ in left middle lobe (*see Fig.* 60).

Liver: Normal.
Gall-bladder: Normal.
Pancreas: Normal.
Kidneys: Normal.
Genital Organs: Normal.
Ductless Glands: Normal.
Spleen: Small, flattened, pale.
Alimentary Canal: No free blood in peritoneal cavity. Stomach and small and large intestines normal.
Bladder: Moderately distended with clear urine, wall normal.
Bones: No fractures of long bones, ribs, or pelvis.
Blood: Fluid.
Central Nervous System: Not examined.

SUMMARY OF PATHOLOGICAL FINDINGS.—Extensive hæmorrhage in both lungs; acute interstitial and subpleural emphysema; pneumothorax; petechiæ in trachea; blood-clot in bronchi and trachea.

Monkey 10.—Chest protected. In sea from 12.50 p.m. to 1.15 p.m. approximately. *In extremis* 11 min. afterwards. Post-mortem examination 3½ hr. later. No signs of external injury. No bleeding from orifices. No hæmorrhage in skin, subcutaneous tissue, or muscle. Eyes normal.

Heart: No pericardial fluid. Pericardium free from hæmorrhages. No subendocardial hæmorrhage. Myocardium, valves, papillary muscles normal. No displacement of heart. No air in cavities. Coronaries filled with blood, normal. Heart in systole. Large blood-vessels normal.

Lungs: Moderate hæmorrhage in right lung, one large hæmorrhage in upper lobe, two massive hæmorrhages in middle lobe, one large and two smaller in lower lobe. A few small hæmorrhages at margins of left lung. Hæmorrhages mainly peripheral, extending about half way towards hilum; diffuse congestion towards hilum. Emphysema diffuse throughout left upper and at periphery of left lower lobe with subpleural bullæ here. A little emphysema near right base only. Pleural cavities free from blood and air. No evidence of pleural injury. No bronchial petechiæ or blood-clot. Trachea contained frothy fluid, not blood-stained. No petechiæ in wall.

Liver: Normal.
Gall-bladder: Normal.
Pancreas: Normal.
Kidneys: Normal.
Genital Organs: Normal.
Ductless Glands: Normal.
Spleen: Subcapsular hæmorrhages on anterior surface. Not distended.
Bladder: Partly distended with clear urine, wall normal.
Alimentary Canal: No free blood in peritoneal cavity, no gas or intestinal contents. Stomach normal. Three small submucous and subperitoneal hæmorrhages in loops of small intestine (about 3 mm. × 2 mm.). One large submucous hæmorrhage in middle of rectum (0·5 cm. × 0·5 cm.).
Bones: No fractures in long bones, ribs, or pelvis.
Blood: Mostly fluid.
Central Nervous System: Brain kept for examination in London.

SUMMARY OF PATHOLOGICAL FINDINGS.—Hæmorrhage and moderate acute emphysema in both lungs; subcapsular hæmorrhages in spleen; small hæmorrhages in small intestine and rectum.

Monkey 11.—Abdomen protected. In sea from 12.50 p.m. to 1.15 p.m. approximately. Found dead. Post-mortem examination 3¾ hr. later. No signs of

external injury. Eyes normal. No bleeding from orifices. No hæmorrhage in skin, subcutaneous tissue, or muscles.

Heart: Pericardial fluid scanty, clear. Pericardium, myocardium, valves, papillary muscles normal. Heart not displaced. No air in cavities. Coronaries filled with blood, normal. Heart in systole. Large blood-vessels normal.

Lungs: Pleural cavities normal. Massive hæmorrhage throughout whole of right lung, affecting all lobes and extending almost to hilum. A few large areas of hæmorrhage in left upper and middle lobes beneath pleura, extending about one-third of the distance towards hilum. Numerous small hæmorrhages (petechiæ and fused petechiæ) in left lower lobe. A few patches of emphysema subpleurally in right and left upper lobes and at right base. Numerous petechiæ in posterior wall of trachea and main bronchi scattered at intervals between cartilage rings for about half the length of the trachea. No blood-clot in bronchi or trachea.

Liver: Normal.
Gall-bladder: Normal.
Pancreas: Normal.
Kidneys: Normal.
Genital Organs: Normal.
Ductless Glands: Normal.
Spleen: Multiple small subcapsular hæmorrhages, not enlarged.
Bladder: Moderately distended with urine; wall normal.
Alimentary Canal: No free blood in peritoneal cavity. Multiple small hæmorrhages scattered throughout wall of small intestine, submucous in situation. Blood-stained intestinal contents close to these. Stomach and rest of intestine normal.
Bones: No fractures of long bones, ribs, or pelvis.
Blood: Mostly fluid.
Central Nervous System: Brain sent to London.

SUMMARY OF PATHOLOGICAL FINDINGS.—Massive hæmorrhages in right lung, moderate hæmorrhage in left lung; bronchial and tracheal petechiæ; multiple submucosal intestinal hæmorrhages with bleeding into small intestines; subcapsular hæmorrhages in spleen.

40 yd. from Depth Charge.—

Monkey 12.—No protection. In sea from 12.51 p.m. to 1.16 p.m. approximately. Found dead. Post-mortem examination after 3¾ hr. No signs of external injury. Eyes normal. No bleeding from orifices. No hæmorrhage in skin, subcutaneous tissue, or muscles.

Heart: No pericardial fluid. Pericardium, myocardium, valves, papillary muscles normal. No displacement of heart. No air in cavities. Coronaries filled with blood, normal. Heart in systole. Large blood-vessels normal.

Lungs: Some diffuse hæmorrhage, not very severe, in right lower and left upper, middle and lower lobes, with small discrete subpleural hæmorrhages (about 0·5 cm. diam.) beneath pleura of right upper and middle lobes. Hilar regions almost unaffected. No petechiæ in walls of bronchi or trachea. No blood in bronchi. No obvious emphysema, certainly no subpleural bullæ. Pleural cavities free from blood or air. No injury of pleuræ. Trachea free from fluid.

Liver: Normal.
Gall-bladder: Normal.
Pancreas: Normal.
Spleen: Normal.
Kidneys: Normal.
Genital Organs: Normal.
Ductless Glands: Normal.
Bladder: Empty, wall normal.
Alimentary Canal: No free blood in peritoneal cavity. Stomach and small intestine normal. Fairly extensive areas of hæmorrhage (subperitoneal and submucous) scattered throughout sigmoid colon. Blood-stained contents in sigmoid colon. Rectum normal.
Bones: No fractures in long bones, ribs, or pelvis.
Blood: Mostly fluid.
Central Nervous System: Brain sent to London.

SUMMARY OF PATHOLOGICAL FINDINGS.—Moderate diffuse hæmorrhage in both lungs; hæmorrhage in wall of sigmoid colon, with bleeding into its lumen.

Monkey 13.—No protection. In sea from 12.51 p.m. to 1.15 p.m. approximately. Found dead. Post-mortem examination after 3¾ hr. No sign of external injury. Eyes normal. No bleeding from orifices. No hæmorrhage in skin, subcutaneous tissues, or muscles.

Heart: Pericardial fluid scanty, clear. No pericardial lesions. Myocardium, valves, papillary muscles, coronaries normal. No displacement of heart. No air in cavities. Heart in systole. Large blood-vessels normal.

Lungs: Pleural cavities free from blood or air. Pleura normal, except where emphysema had involved subpleural tissues; a few bullæ here. Two small hæmorrhages (about 0·5 cm. diam.) close to right and left apices; small hæmorrhages scattered throughout right upper and middle lobes and left upper, middle, and lower lobes; severe subpleural hæmorrhage at right base. A few medium-sized hæmorrhages (about 1 to 2 cm. diam.) at left base. Some diffuse emphysema at right and left apices and at left base, involving bronchi and interlobular septa, reaching to pleura in places. No blood in bronchi or trachea, no petechiæ in their walls. Frothy fluid in trachea.

Liver: Normal.
Gall-bladder: Normal.
Genital Organs: Normal.
Adrenals: Normal.
Thyroid: Normal.
Pancreas and *Thymus*: Petechiæ.
Kidneys: Capsular hæmorrhage in right and left kidneys, more marked in right; remainder of organs normal. No bleeding into pelvis. Ureters normal.
Spleen: Multiple subcapsular hæmorrhages. Collapsed pulp.
Bladder: Partly distended with clear urine. Wall normal.
Alimentary Canal: Extensive submucous and adventitial hæmorrhages in wall of stomach, and small and large intestines. Gut distended at site of hæmorrhages, some of which were 2 to 3 cm. in length. Gut contents blood-stained, with mucus and bright red blood; some tearing of mucosa, but no perforation. No free blood or gas in peritoneal cavity (*see Fig.* 64).
Bones: No fracture in long bones, ribs, or pelvis.
Blood: Fluid.
Central Nervous System: Brain sent to London.

SUMMARY OF PATHOLOGICAL FINDINGS.—Hæmorrhage and acute emphysema in both lungs; petechiæ in pancreas and thymus; subcapsular hæmorrhages in spleen and kidneys; multiple large hæmorrhages in stomach and small and large intestines, with bleeding into bowel.

45 yd. from Depth Charge.—

Pig 1.—No protection. In sea from 12.53 p.m. to 1.18 p.m. Killed 11 min. after exposure to depth charge. Post-mortem examination about 4 hr. later. No signs of external injury. Eyes normal. No bleeding from orifices. No hæmorrhage in skin, subcutaneous tissues, or muscles. Well-nourished animal, with thick abdominal and thoracic walls and a good layer of fat (at least 1 in. thick).

Heart: A little clear pericardial fluid. Pericardium, myocardium, valves, papillary muscles normal. No air in cavities. No displacement of heart. In systole. Large blood-vessels normal.

Lungs: Moderate-sized hæmorrhages (about 0·5 to 1 cm. diam.) at margins of right upper, middle, and lower lobes, and left upper lobe, with scattered petechiæ some little distance in from pleura in all lobes. Hilar regions free from change. No petechiæ in walls of bronchi or trachea, no blood-clot in these. No obvious emphysema. Pleural cavities free from blood or air.

Liver: Normal.
Gall-bladder: Normal.
Pancreas: Normal.
Spleen: Normal.
Kidneys: Normal.
Genital Organs: Normal.
Ductless Glands: Normal.
Bladder: Contained a little clear urine, wall normal:
Alimentary Canal: No free blood in peritoneal cavity. Stomach and small and large intestines normal.
Blood: Still fluid.
Bones: No fractures in long bones, ribs, or pelvis.
Central Nervous System: Not examined.

SUMMARY OF PATHOLOGICAL FINDINGS.—Slight hæmorrhage in both lungs, chiefly subpleural.

Pig 2.—No protection. In sea from 12.53 p.m. to 1.18 p.m. approximately. Killed 11 min. after exposure to depth charge. Post-mortem examination about 4 hr. later. No signs of external injury. No hæmorrhage in skin, subcutaneous tissues, or muscles. Eyes normal. No bleeding from orifices. Well-nourished animal, with thick abdominal and thoracic wall and good layer of fat (at least 1 in. thick).

Heart: A little clear pericardial fluid. Pericardium, myocardium, endocardium, valves, and papillary muscles normal. No air in cavities. No displacement of heart. In systole. Large blood-vessels normal.

Lungs: Small hæmorrhages (up to 0·5 cm. diam.) scattered throughout all lobes, but chiefly situated along margin and at apices. No obvious emphysema. Bronchi and trachea normal, with no blood-clot in their lumen. Pleural cavities free from blood or air. Pleuræ normal.

Liver: Normal.
Gall-bladder: Normal.
Pancreas: Normal.
Spleen: Normal.
Kidneys: Normal.
Genital Organs: Normal.
Ductless Glands: Normal.
Bladder: Empty, wall normal.
Alimentary Canal: No free blood in peritoneal cavity. Stomach and small and large intestines normal.
Blood: Still fluid.
Bones: No fractures in long bones, ribs, or pelvis.
Central Nervous System: Not examined.

SUMMARY OF PATHOLOGICAL FINDINGS.—Small hæmorrhages, chiefly subpleural and apical, in both lungs.

FIBROUS STRICTURE OF THE SMALL INTESTINE FOLLOWING STRANGULATED HERNIA

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FOLLOWING the reduction of a strangulated but viable loop of intestine complete resolution may not occur. In certain circumstances a fibrous stenosis forms which may later cause intestinal obstruction.

There are several types of mechanical obstruction that may arise when a segment of strangulated bowel has been returned to the abdomen. Usually this complication is noted immediately afterwards, for the signs and symptoms of obstruction persist. In comparatively few cases the onset is delayed. Moreover, there are two main types of late intestinal obstruction. One is an *extrinsic* type, in which the serous layer only is involved as the result of a plastic peritonitis. The other is an *intrinsic* type, due to a fibrous stenosis which involves all layers of the gut. In the former, loops of intestine become twisted and kinked, bound to each other and to the abdominal wall or stenosed by encircling adhesions. This form is found also associated with inflamed and incarcerated hernias. The latter, or intrinsic, form is only found after strangulation (on which it is dependent), and it alone is the subject of this communication.

Three recent cases of this intrinsic type of fibrous stricture are reported here, and a brief account is given of its history, pathology, and clinical aspects.

HISTORICAL

The surgeons of the eighteenth century saw many examples of intestinal obstruction following reduction of strangulated hernias by taxis or by operation. Excellent accounts are given by Garengeot (1748) and Arnaud (1748). Richter (1788) warned his readers that even after the happiest cures the patient was not out of danger. Vincent (1781), to whom the credit of first describing this stricture has been given, had seen in the Hanover War a soldier die of obstruction twenty-four hours after an operation for strangulated hernia. A post-mortem examination revealed a stricture of the segment of intestine that had been strangulated. None of these authors, however, describe uncomplicated cases of late stenosis due to a fibrosis which needs at least several weeks to develop.

The first example of an undoubted fibrous stricture is given by Guignard (1846), and from his time the condition was known to French surgeons, Allaux (1860), Anger (1865), Nicaise (1881), and others. Cotte and Leriche (1905) reviewed 39 cases that had then been fully reported, and more recently Delvaux (1932) has added his observations. In Italy Costa reported a case in 1894 and later (1934) described 8 cases he had successfully treated.

In Germany the earlier reported cases appear to have been overlooked. Garré (1892) described a case which stimulated great interest in the condition. His patient, a man aged 27, had a strangulated hernia containing a loop of small intestine 20 cm. long. The gut appeared quite viable after division of the constriction and it was returned to the abdomen. From the third to the tenth day after this operation the patient complained of colicky abdominal pains and passed frequent 'typhoid' stools, but otherwise made an uneventful recovery. His pains returned, however, and nine weeks after his herniotomy he was operated on for intestinal obstruction. Several adherent coils of small intestine were resected and in their midst was a tubular stricture 7 cm. long which barely admitted a probe. Garré considered that venous stasis in the mucosa had led to necrosis and shedding of the epithelium (as shown clinically by the profuse diarrhœa), and that subsequent infection of the intestinal layers resulted in the fibrous stricture. Maas (1895) described another case, and was able to produce a similar stenosis experimentally in rabbits. The condition in Germany is known as 'the intestinal stenosis of Garré' and several of his pupils published other examples of the same stricture and described them at length. Meyer (1905) and Matti (1911) made extensive reviews of the reported cases and investigated the effect of strangulation on the different layers of the intestinal wall.

One of the first cases described in the English literature was by Thompson (1897), from Edinburgh. His patient came to him in an advanced stage of intestinal obstruction twelve weeks after an operation for a strangulated hernia. A loop of bowel containing two fibrous strictures (representing the constriction rings of the original strangulated hernia) was exteriorized and drained by a small Paul's tube. Eighteen days later a resection and anastomosis over a Murphy's button was successfully performed.

Treves (1899) found several cases in London museums and 11 cases recorded in the literature. Subsequently very few cases have been reported by English or American surgeons. At the London Hospital Barnard (1910) found only 1 case in thirteen years, and no case has been recorded in the Bernhard Baron Institute of Pathology till this year.

CASE REPORTS

Case 1.—F. C., 10464/41, a man aged 32, was admitted to the London Hospital on Jan. 11, 1941, with a large strangulated right inguinal hernia. He was operated on by Mr. J. W. Hannay three and a half hours after the onset of his symptoms. The sac was found to contain two loops of small intestine; one loop was 18 in. long, swollen, deep purple, and had a thick œdematous mesentery; the other was 6 in. long and much less affected. As soon as the constriction ring was divided colour returned, peristaltic movements passed freely over both loops, and the vessels in the mesentery and gut wall pulsated normally. Both loops were returned to the abdomen and a repair was performed.

The patient recovered well, and three weeks after his operation he was discharged to a convalescent home. For several days before leaving hospital his bowels were open as many as five times a day and while at the convalescent home he had an attack of colicky abdominal pain associated with vomiting. Subsequently he had periodic attacks of similar type, approximately once a week, which were relieved by vomiting. His bowels were open at least twice a day. The motions were loose and offensive; they often contained slime, but never blood. Although he was always a lean man, he lost 2 st. in four months following his operation.

In spite of these attacks he did not come to hospital till May 5, 1941, four months after his hernia had been reduced. He had been vomiting fæculent matter almost continuously for the previous twenty-four hours. He was pale, wasted, and dehydrated. An enema produced a pale stool but no flatus, and when repeated there was neither fæces nor flatus. His stomach was kept aspirated by means of a Ryle's tube and an intravenous drip saline with 5 per cent glucose was started.

Ten hours after his admission to hospital his abdomen was opened under local anæsthesia (H. C. B.). A tubular stenosis of the ileum about 6 in. long was found 3 ft. above the ileocæcal junction. There was no kinking of the gut and no adhesions. The stenosed area was indurated and puckered and presented a reddish-purple colour. There were several enlarged hard glands in the adjacent mesentery. Owing to the enormous distension and hypertrophy of the proximal coils of small intestine it was considered unwise to attempt a lateral anastomosis and an ileostomy was performed above the constriction. The diagnosis of regional ileitis was made.

The patient improved rapidly and was maintained on intravenous drip therapy for the next seven days. His abdomen was then reopened under gas, oxygen, and ether anæsthesia (H. C. B.) and a lateral anastomosis performed to short-circuit the obstruction. Unfortunately there had been some leakage from his ileostomy which had led to localized peritonitis. He recovered well at first and his bowels moved normally, but two days after the operation he collapsed suddenly and died within seven hours.

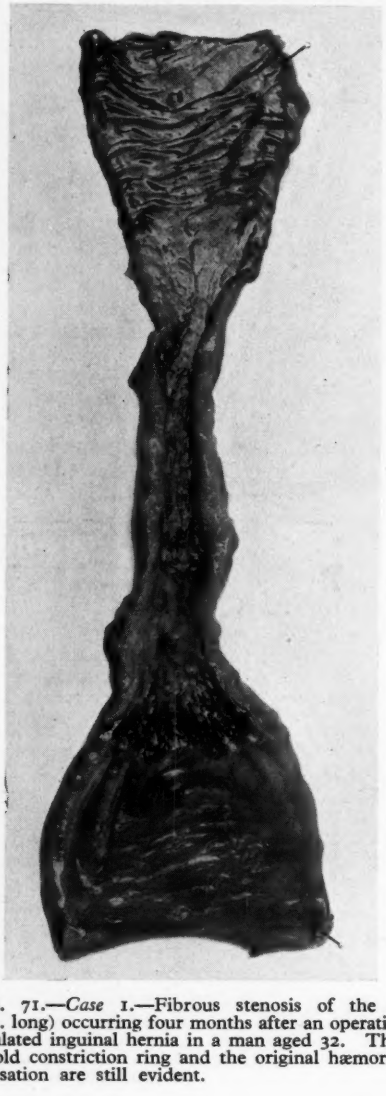

FIG. 71.—*Case* 1.—Fibrous stenosis of the ileum (14 cm. long) occurring four months after an operation for strangulated inguinal hernia in a man aged 32. The site of an old constriction ring and the original hæmorrhagic extravasation are still evident.

SUMMARY OF NECROPSY (P.M. 56/1941).—Small pulmonary embolus. Thrombosis in femoral vein. Bronchopneumonia. Intestinal obstruction. Constriction of small gut by scarring, following strangulation in hernial sac. Recent operations—ileostomy followed by ileo-ileal anastomosis. Old operation—reduction and radical cure of strangulated inguinal hernia.

Great stenosis of portion of small intestine (14 cm. long) 65 cm. above cæcum, its serous surface being wrinkled, the lumen measuring from 2 cm. to 2·5 cm. in circumference, its wall being moderately thickened, the velvety lining being grey and stippled with black streaks of hæmorrhage, its upper end being sharply demarcated as a distinct, thin transverse scar bordered below by short longitudinal ridges, its lower end showing no distinct line of demarcation from the gut below it. No peritoneal adhesions over the stenosed gut. Only one area of stenosed gut found. No evidence of the other loop said to have been strangulated in the hernia. Intact recent healing lateral anastomosis uniting ileum at a point 26 cm. above cæcum to ileum at a point 93 cm. above cæcum, the stoma being 3·5 cm. long. No dilatation of small intestine below stoma (from 4 cm. to 6 cm. circumference), nor of large intestine, the transverse colon being 6 cm. circumference. Considerable dilatation of small intestine above stoma (from 8 cm. to 10 cm. circumference). Great submucous œdema and organizing fibrinous peritonitis of gut for 60 cm. above stoma. Few patches of fibrinous peritonitis scattered in rest of abdomen, thin translucent pink fluid (7 oz.) in peritoneal cavity. Lightly adherent thrombotic embolus (3·5 cm. × 1 cm.) in pulmonary artery in lower lobe of right lung, small piece of similar thrombus in left femoral vein. Œdema of lungs, purulent bronchiolitis, moderately developed bronchopneumonia. Brown atropy of heart (6·75 oz.). Slight œdema of legs. Great œdema of scrotum. Very wasted man. (*Fig.* 71.)

Microscopical Examination.—From widely separated parts of the stenosed area of gut four pieces were cut longitudinally and two transversely to include all the circumference. There are small scattered remnants of mucosa in the two pieces taken from the lower quarter of the area. In the four sections from other parts of the area the mucosa is absent and the innermost layer is a very cellular granulation tissue consisting of capillaries and fibroblasts together with very numerous plasma cells, lymphocytes, and large histiocytes. There is a lesser degree of similar inflammatory infiltration in the adjacent zone, which is chiefly composed of bundles of muscle and has evidently developed as the result of hyperplasia of the muscularis mucosæ; the muscle-fibres are separated from one another by a delicate reticulum of collagen and elastic fibres. In the submucosa there is dense fibrosis. The inner circular and outer longitudinal coats of the muscularis are hypertrophied; the connective tissue septa between their bundles of muscle are very slightly thickened by dense fibrosis. The subserous connective tissue is a slightly thickened band of dense fibrous tissue and is separated by a conspicuous thin layer of elastic fibres from the superficial zone of organizing fibrinous peritonitis. The thickened fibrous tissue in the submucosa, muscularis, and subserosa is rich in fibroblasts, and in places also in plasma cells, lymphocytes, and large histiocytes. Nowhere in the sections is there any pigment remaining from old hæmorrhage.

Case 2.—M. J., a man aged 78, was operated on May 7, 1941, for a strangulated right inguinal hernia by Mr. R. Parkinson. For years he had had a right inguinal hernia which was controlled by a truss, but for twelve hours it had been irreducible and painful, and repeated attempts at taxis had failed. The sac contained a segment of gut 8 in. in length and an œdematous mesentery. There was a diffuse, extensive hæmorrhage beneath the serous layer and in the mesentary. This was considered to be due to the attempts to reduce the hernia by taxis. As peristaltic waves passed over the whole segment of the affected loop of

bowel once the obstruction was relieved, it was considered to be viable and was replaced in the abdomen. A radical cure was performed and he made an uneventful recovery.

He was readmitted with intestinal obstruction five weeks later (June 15). He stated that he had had some mild attacks of colicky abdominal pain soon after his operation, but for the last twelve days they had become increasingly frequent and severe and for six hours he had been vomiting brown fluid.

At operation (Mr. Henry Thompson) an indurated and constricted knuckle of gut was found fixed in the region of the old herniorrhaphy and causing complete obstruction. Three feet of intestine including the constriction were resected and an end-to-end anastomosis performed. The patient made an uninterrupted recovery.

PATHOLOGIST'S REPORT.—

Macroscopical Description (*Fig.* 72).—The specimen (S.D. 321/1941) is a portion of small intestine (25 cm. long). Near its middle there is a conspicuous kink, forming an angle of about 15°, the space in the angle is filled with dense unbreakable fibrous adhesions and the mesentery here is indurated by indistinct white fibrous strands permeating its fat. At the kink the lumen of the gut is greatly narrowed, being only 2 cm. in circumference, the mucosa is opaque and white, the wall is greatly thickened (0·7 cm. thick) for a distance of 6 cm. The gut above the kink is dilated (8 cm. circumference) and hypertrophied (muscularis 0·25 cm. thick), while below this stenosed area the circumference is 4·5 cm. and the muscularis is so thin as to be difficult to measure accurately.

Microscopical Examination.—Microscopical sections were made of two large pieces from the stenosed area. Over a large part of one piece all epithelial elements of the mucosa have disappeared and the mucosa is replaced by a pyogenic granulation tissue consisting of a superficial fibrino-purulent exudate and an underlying granulation tissue composed of very numerous lymphocytes, plasma cells, large histiocytes and dilated capillaries, and a few fibroblasts. The muscularis mucosæ has been destroyed and thus the mucosal granulation tissue has blended with a similar but more fibrotic inflammation in the submucosa. The muscularis is disintegrated by severe inflammation in a small area where it has been exposed by ulceration. Throughout the rest of the two pieces the architecture of the mucosa is preserved, but in it and all other coats there is severe inflammation, which is associated with extensive bands of dense fibrosis in the subserous connective tissue and with much less fibrosis in the muscularis and submucosa.

Case 3.—K. G., a woman of 59, was operated on at Whipps Cross Hospital on Feb. 6, 1940, for a strangulated right femoral hernia. The sac contained a dark loop of bowel which was considered to be viable. It was returned to the abdomen and a radical repair was performed.

Eleven days later she was operated on at the same hospital for acute intestinal obstruction. The obstruction was located in the small intestine, apparently at the site of the previously strangulated loop. The gut here had the appearance of an extensive inflammatory œdema. The circulation in the mesentery was normal. An ileostomy was performed above the obstruction. The patient's symptoms were temporarily relieved and she was discharged a month later.

Spasms of colicky abdominal pain, however, soon returned and gradually increased in severity. Her bowels were open twice a day and the motions were loose. Three months after her first operation she was admitted to Aldersbrook Hospital and was operated on by Mr. Henry Thompson. The gut (at the site of the old ileostomy) was found adherent to the abdominal wall. Proximal coils of ileum going to this segment were distended, whereas those distal

FIG. 72.—*Case* 2. Fibrous stenosis of the ileum causing intestinal obstruction five weeks after an operation for strangulated inguinal hernia in a man aged 78.

to it were collapsed. The segment was resected and an end-to-end anastomosis performed. The patient made an uninterrupted recovery.

The resected portion of bowel was not kinked, but contained a well-marked fibrous stenosis of the intrinsic type (*Fig.* 73).

Commentary.—These three cases illustrate many features of this condition. In *Case* 1 there is an exceptionally long tubular stricture. Even so it represents all that remains of the original strangulated loop 18 in. long. The diffuse fibrosis

has caused both a circular and longitudinal contracture. The original constriction rings and the hæmorrhagic extravasation are still clearly visible (*Fig.* 71).

In *Case* 2 (*Fig.* 72) there are both extrinsic and intrinsic factors causing obstruction. The adhesions to the old herniorrhaphy site and the kinking of the gut are the result of a plastic peritonitis; the fibrous stricture represents part of the old strangulated hernia. These two factors are commonly associated.

FIG. 73.—*Case* 3. Fibrous stenosis of the ileum causing intestinal obstruction three months after an operation for strangulated femoral hernia in a woman aged 59.

In *Case* 3 the stricture has been observed during its development. At first is was thought to be an 'inflammatory œdema' which would subside without causing any permanent stenosis. This area has, however, healed by scar tissue and a stricture has formed which probably represents the whole of the original strangulated loop (*Fig.* 73).

AETIOLOGY

When a loop of intestine becomes strangulated, the mucosa (where the blood-vessels terminate) is first and most seriously affected. Early observers noted this and it can be easily confirmed experimentally. Hoffmann (1907) showed that even at the constriction rings, in spite of the direct pressure on the serous coat, the mucosa always suffers most. Venous congestion is followed by thrombosis of the vessels, hæmorrhages, and necrosis of the epithelium. However, the mucosa has remarkable powers of regeneration, and in spite of this hæmorrhagic necrosis it is usually completely and rapidly restored. In a few cases complete resolution does not take place and widespread fibrosis occurs, resulting in healing by scar tissue. When this happens probably there is not only extensive necrosis of the mucosa but severe and permanent damage to the submucosal layers as well. Moreover, here, as in other parts of the body, there is a tendency to form fibrous tissue wherever there is a large extravasation of blood. Once the epithelium is destroyed the deeper layers are open to infection from the intestinal tract. This may hinder the normal healing processes, though it must be remembered that in typhoid fever complete regeneration regularly occurs. The stricture that forms after strangulated hernia is comparable to the cicatrices in an appendix which has been the seat of previous attacks of appendicitis. Here the same factors of thrombosis and epithelial necrosis often coexist.

The clinical signs of viability in a loop of bowel which has been strangulated give very little information regarding the state of the mucous membrane. The gut may regain its tone and colour, peristaltic waves return, and the blood-vessels may pulsate with apparently increased vigour, and yet there may be widespread damage to the mucosa. Hæmorrhagic necrosis of the epithelium is indeed very common after strangulation. It is therefore difficult to explain why this cicatricial stenosis should so rarely form. In all the reported cases the gut was considered to be viable according to the usual clinical criteria. Several observers (Costa, Delvaux), however, have noted (and this observation is confirmed in the first two cases reported here) that at the initial operation there was an extensive subserous and mesenteric hæmorrhage affecting the loop of bowel and its mesentery. These signs indicate a widespread thrombosis and hæmorrhagic infiltration throughout all the layers of the intestinal wall. They suggest that the obstruction was more slowly progressive than is usual, or that there had been early venous thrombosis in the mesenteric vessels following the onset of strangulation.

PATHOLOGY

This stricture has been described following the reduction of a strangulated hernia in men and women both young and old. No particular type of hernia predisposes to the condition, nor can any conclusions be drawn from the length of time that has elapsed before the strangulation has been relieved. It has, moreover, been observed following reduction of a strangulated hernia by taxis (Baisch, 1904; Cotte and Leriche, 1905; Costa, 1934), without operation. Most of the strictures have been seen in the ileum, several in the jejunum, but none in the large intestine. The German authors in particular have stressed the importance of a prodromal phase during convalescence. There are two characteristic symptoms, intestinal hæmorrhage and diarrhœa. Hæmorrhage during the first twenty-four hours is due

to trauma and has no special significance. Should it occur several days after the reduction of the hernia it probably indicates sloughing of necrotic areas of epithelium. Diarrhœa is a more constant sign. It usually appears about the third day after operation and may last up to a fortnight. The stools are fluid and profuse. They are similar to those commonly seen in typhoid fever, and are caused by the ulceration of the necrotic epithelium.

The fibrosis that results causes a progressive stenosis. After a varying latent interval symptoms of intestinal obstruction arise. Usually they are first noticed within a few weeks of the reduction of the hernia, but they may not arise for months or even years afterwards. Baisch (1904) described a case which caused obstruction fourteen years later. The picture is one of chronic progressive obstruction of the small gut. Attacks of colicky abdominal pain become increasingly frequent and severe. There is a steady loss of weight and a progressive anæmia. This may be of macrocytic type (Ginzburg and Klein, 1928), as has been noted in small-gut obstruction from other causes.

Not all cases, however, proceed so far. The lesion has been noted in the course of laparotomies for other surgical conditions (Delvaux, 1932), and at routine post-mortem examinations (Haasler, 1903; Lejars, 1912) of patients who had had a strangulated hernia some years previously, but had suffered no symptoms from the stricture which followed.

TREATMENT

There is little to suggest to the surgeon at the time of the operation for the strangulated hernia that a fibrous stenosis may follow. We have seen that an intact serous coat with normal peristaltic waves and pulsation in the vessels is an unreliable guide to the state of the mucosa. When the gut wall is thin there is less difficulty than when it is swollen. Once the obstruction to the blood-vessels has been relieved the gut either regains its tone and colour quickly or remains soft, dark, and obviously non-viable. When, on the other hand, the gut and its mesentery are swollen, indurated, and hæmorrhagic, and this induration persists after the strangulation has been relieved, there is a possibility of subsequent fibrosis and a stricture forming. However, the complication is so rare that provided the gut is viable according to the usually accepted clinical standards there is no indication for resection at this stage.

Diarrhœa and intestinal hæmorrhage during convalescence also suggest the possibility of a stricture forming, though usually they subside without further complications developing. Should signs of obstruction follow later, and especially when preceded by these prodromal features, a fibrous stenosis may be suspected. The stricture is slowly progressive and laparotomy should be performed without undue delay. Most cases have been successfully treated by either a short-circuiting lateral anastomosis or a resection of the affected loop and anastomosis.

SUMMARY

Three cases are reported of late intestinal stenosis following strangulated hernia.

Occasionally, although a strangulated loop of intestine is viable, as shown by the return of its colour, pulsation in its vessels, and peristaltic waves, there is such extensive damage to the inner layers that complete resolution cannot take place. Fibrosis occurs either at the site of the constriction ring or throughout the length of the strangulated intestine, and leads to the formation of an annular or a tubular stricture. This is especially likely to occur when there has been an extensive hæmorrhagic infiltration of the strangulated intestine and its mesentery.

Clinically, a few days after the operation, diarrhœa with blood in the stools is sometimes noticed. Weeks, months, or even years later the signs of a slowly progressive small-gut obstruction develop, and at laparotomy a fibrous stenosis at the site of the old strangulated loop is found.

I am indebted to Mr. Henry Thompson, who has kindly allowed me to report 2 cases he has successfully treated; to Dr. W. W. Woods for the pathological reports; to Mr. Alan Perry for permission to publish the first case; and to Dr. Coleman, Medical Superintendent of Aldersbrook Hospital, for permission to publish the second and third cases.

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TRAUMATIC RUPTURE OF THE URETHRA

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RUPTURES of the urethra usually occur in the male. They are described as anterior, in front of the triangular ligament—usually in the bulb and rarely in the penile urethra; or posterior, behind the triangular ligament, in the membranous or prostatic urethra, and usually at the junction of these two divisions. The latter are often associated with a fracture of the pelvis. Ruptures may be complete, involving the whole circumference; incomplete, involving a segment only; or the damage may involve the mucosa only. They are much more frequent in young adults and, in so far as the consequences of indifferent treatment or lack of it may be serious from the point of view of discomfort or even be a menace to health, it is important that treatment should be effective and instituted early.

Urethral ruptures are, however, rare; few cases occur in the practice of an individual surgeon, and for that reason it is considered of interest to put on record 3 personal cases of complete rupture of the anterior urethra, the bulbous portion. An extensive review of the literature has recently been made by Simpson-Smith (1936). To these cases may be added further records, often without much detail as to type and end-results, made amongst others by Hamilton Bailey (1928) 4 cases, 3 anterior and 1 posterior; Higgins (1930) 12 cases, of which 7 were complete anterior; O'Connor (1936) 31 cases, of which 8 were due to a fall astride a manhole cover, this coming up and hitting the perineum; Gayet (1936) 3 cases; Culver (1938) 32 cases, of which 14 were bulbous and 18 posterior urethral; Ockerblad and Carlson (1938) 5 cases; Ball (1938) 5 cases, of which 1 was anterior and 4 posterior; Hutchins (1938) 10 cases, of which 6 were bulbous and 4 posterior; Moore (1938) who collected 33 cases in ten years, 10 being incomplete ruptures, and of which 13 were posterior and 20 anterior, at the Royal Melbourne Hospital, these cases being attended by 13 surgeons; and Harrison (1941) 21 cases, of which 12 were bulbous and 9 membranous.

CASE REPORTS

Case 1.—J. H. P., male aged 29, a mill hand, admitted to the Birkenhead General Hospital on Aug. 6, 1935, with a history of falling astride a board.

On admission there was considerable bleeding from the urethra, retention of urine, and a hæmatoma in the perineum. The house surgeon attempted to pass a catheter but failed. I saw the patient within an hour or two and he was taken to the theatre. Under spinal anæsthesia, a further attempt at catheterization being unsuccessful, a suprapubic cystostomy was done and a bougie passed into the internal meatus. The patient was put into the lithotomy position and a midline incision was made in the perineum, a bougie having been passed down the anterior urethra. The two ends of the urethra were thus easily visualized. There was a great deal of bleeding which was controlled in part by pressure. It was found impossible to bring the two ends of the urethra together even after straightening out the lower limbs. No attempt was made to mobilize the anterior urethra. A gum-elastic catheter was passed through the anterior urethra into the proximal urethra and the perineal wound was lightly packed with vaseline gauze. A suprapubic tube was inserted and the patient sent back to bed.

He was nursed in the Trendelenburg position; the suprapubic tube was removed in four days and

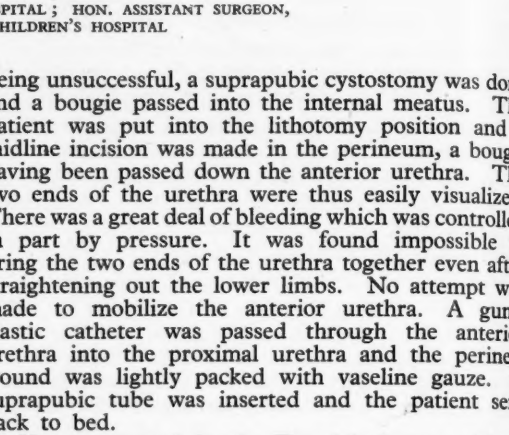

FIG. 74.—*Case* 1. Urethrogram showing narrowing of the bulbous urethra.

replaced by a Hamilton-Irving apparatus. There was no drainage from the indwelling catheter and no gross infection. The catheter was removed in six days and a second gum-elastic catheter inserted. This produced a mild urethritis and was removed in forty-eight hours. The patient was kept constipated for five days; the pack was removed in forty-eight hours and the perineal wound irrigated three times daily with eusol.

A bougie was passed gently down the urethra a fortnight after operation under anæsthesia and rather to my surprise went in quite easily. This was repeated in forty-eight hours and thereafter a bougie was passed daily. The perineal wound after healing broke down temporarily and a catheter was inserted for forty-eight hours. Both suprapubic and perineal wounds were healed firmly and he was passing urine well by Sept. 9, five weeks after the operation. The patient was discharged on Sept. 18.

He attended the out-patient department and had bougies passed weekly for a month, and then fortnightly for a further month, and subsequently every

two months, followed by visits every six months. Urethroscopy in December, 1935, showed scarring on the floor and right wall, with a fold coming down from the roof and left wall. He took an 11/14 Lister bougie with ease. He was seen last in April, 1938, six months after his previous visit, when an 11/14 bougie passed with great ease. The urethrogram showed some narrowing of the bulbous urethra (*Fig.* 74).

Case 2.—P. B., male aged 42, a labourer, was admitted to the Birkenhead General Hospital on Dec. 1, 1935. Whilst carrying timber one leg fell into a pit and his perineum caught on the edge. There was bleeding from the urethra, with a slight hæmatoma in the perineum and retention of urine. Attempt by the house surgeon at catheterization failed. I saw him four hours later, when his bladder was up to his umbilicus. I operated under spinal anæsthesia; this did not produce extravasation. The bladder was opened suprapubically and a bougie was passed into the internal meatus. Another bougie was passed down the anterior urethra and the perineum incised in the midline. The two ends of the torn urethra were visualized and it was possible to bring them together. I passed four sutures into the roof of the urethra and two into the floor, the edges coming into apposition easily. A catheter was passed along the urethra for thirty-six hours; the perineal wound was packed and the bladder drained through the suprapubic wound. This patient was incontinent of fæces on the theatre table. The nursing in this case was similar to that in the first, except that no catheter was inserted after the removal thirty-six hours later of the one inserted in the theatre. A bougie was passed ten days after operation and then on alternate days. The patient was discharged from hospital on Dec. 23, three weeks after admission, with both wounds soundly healed and passing urine normally. Bougies 10/13 were passed at weekly intervals with ease. Urethroscopy in February, 1936, showed no evidence of stricture and the urethrogram shows a wide urethra (*Fig.* 75). Bougies were passed every four to six months subsequently. This patient complained of a swelling in the perineum on working and defæcation —but nothing could be made out clinically apart from separation of perineal muscle-fibres. He was last seen in November, 1939, twelve months after his previous attendance, and an 11/14 bougie was passed with great ease.

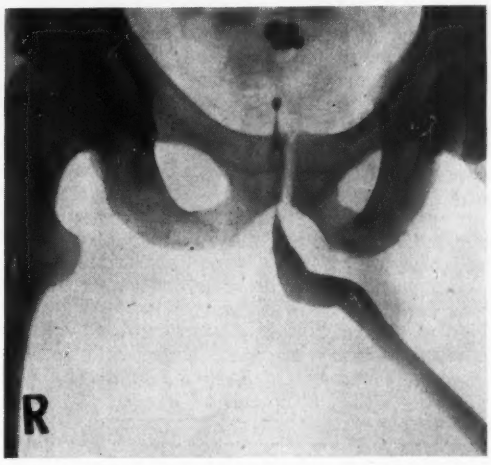

FIG. 75.—*Case* 2. Urethrogram showing a wide urethra.

Case 3.—W. M., male aged 53, plater, was admitted to the Birkenhead General Hospital on Sept. 10, 1936. He had fallen from a height of 10 ft. astride a spar of a trestle. There was retention of urine, bleeding from the urethra, and a hæmatoma in the scrotum and perineum. There was tenderness also over the pubic ramus. Attempt by the house surgeon at catheterization had been made unsuccessfully. I operated four hours later, opening the bladder suprapubically and then incising the perineum. There was considerable bruising in the perineum in addition to the marked bleeding. In this case I sutured the roof only—and passed a catheter into the bladder, the rest of the procedure being similar to that in the previous cases.

In this case, however, though the patient was also nursed in the Trendelenburg position, the

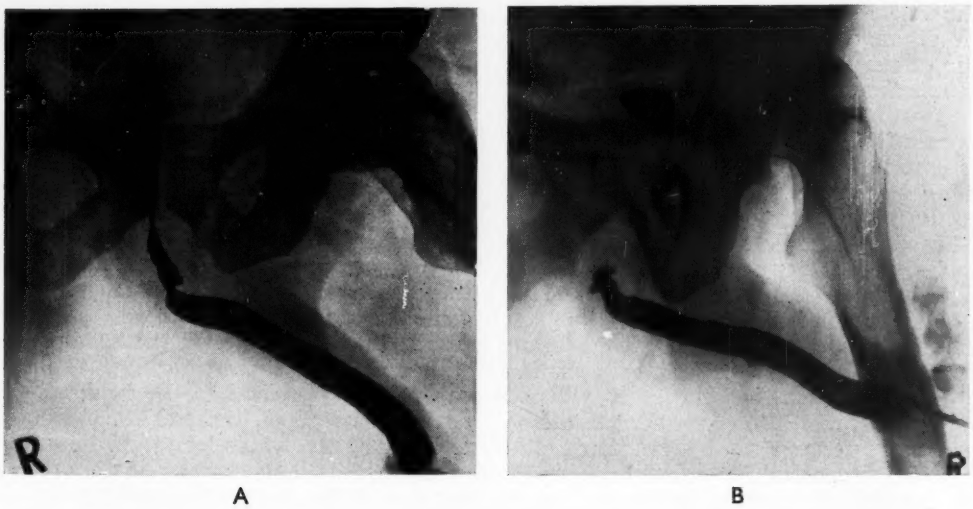

FIG. 76.—*Case* 3. A, Urethrogram taken Nov. 28, 1936, showing narrowing in the bulb; B, Urethrogram taken April 16, 1941, showing a practically normal urethra.

indwelling catheter drained urine and he developed urethritis, so the catheter was removed after five days. It is likely that whereas in the previous two cases the indwelling catheter had been lying just short of the internal sphincter, in this case the eye of the catheter was in the bladder and despite the foot of the bed being elevated, urine drained down the catheter. On Sept. 22 bougies were passed in the theatre—some difficulty was experienced, but no anæsthetic was given. Bougies were passed again on Sept. 30, and subsequently weekly.

The patient was discharged on Oct. 14, five weeks after operation, with both wounds healed well and passing urine normally. This patient had a fracture of the descending pubic ramus. He attended at weekly intervals and dilatation from 6/9 to 9/12 was carried out, there being a definite stricture. This was confirmed on urethroscopy and a urethrogram showed definite narrowing of the lumen (*Fig.* 76, A). Bougies were passed weekly for a further two months and then fortnightly, the interval gradually increasing and the urethra taking a 12/15 bougie easily by March, 1937. Since this date he has been attending at three-monthly and then at six-monthly intervals. He was seen in December, 1940, when a 12/15 bougie was passed easily, and again in April, 1941, when the bougie was once more easily passed. A urethrogram taken at this date shows an almost normal urethra (*Fig.* 76, B). He was seen again in April, 1942, when a 12/15 bougie was passed easily. This patient complained of impotence for a few months after his discharge from hospital.

DISCUSSION

Treatment of ruptures of the urethra is by no means standardized, but a review of the literature tends to show that though differing in detail, the majority of surgeons subscribe to certain principles of treatment. These principles may be briefly stated as: (1) The deviation of the urine from the traumatized area; (2) The reconstruction of the urethral channel; (3) The maintenance of the calibre of the urethra during healing and during a variable post-operative period.

On the whole there is more unanimity in the treatment of ruptures of the posterior urethra than those of the anterior urethra. The former are often associated with fractures of the pelvis and usually with considerable shock. Only the minimal operative intervention is usually possible, but it is imperative to prevent or correct the backward displacement of the posterior urethra which occurs due to the rupture of the pubo-prostatic ligaments and the weight of the prostate. A suprapubic cystostomy is done and a catheter is threaded across the torn urethra. This indwelling catheter acts as a urethral splint and should not be used for drainage. To prevent the latter it may be necessary to apply suction drainage to the suprapubic tube. It will be noted that this operation also allows the hæmatoma in the space of Retzius to be evacuated and drained. If it is impossible to thread the catheter along the ruptured urethra bridging the gap, a tube should be passed through the posterior urethra into the perineum where it is cut down on—thereby fixing the distal end of the posterior urethra (Hamilton Bailey, 1928). Unfortunately contrary to common teaching, strictures do occur in the posterior urethra (Simpson-Smith, 1936; Ball, 1938). Simpson-Smith considered that possibly these were due to sepsis carried to the site by threading a catheter along the anterior urethra. He suggested that the catheter be passed in the reverse direction, and perhaps this would diminish the chances of subsequent stricture formation. Possibly the use of the sulphanilamide group of drugs may diminish the sepsis.

Because of the poor general condition of the patient the treatment advocated above is all that is possible, though some surgeons believe that in posterior urethral ruptures the perineum should be incised and the ends of the urethra united, thereby minimizing the possibility of stricture formation. With the present active treatment of shock the more extensive procedure may be feasible.

Complete anterior urethral ruptures may be dealt with in one of three ways :—

1. Suprapubic cystostomy only, the urethra not being interfered with. After 6 to 8 weeks, when the perineal hæmatoma has settled down, a formal repair of the ruptured urethra is carried out. This treatment is in accord with the modern tendency not to carry out any reparative procedure in the presence of acute trauma for fear the onset of infection vitiates the result. This tendency, for instance, in modern orthopædics, has been carried too far, and many cases which could have been dealt with by primary suture are left. In cases of anterior urethral rupture not only does secondary repair incur two operations, but a longer period of convalescence—spent with a suprapubic cystostomy—and the secondary operation may be difficult. If an error of diagnosis has been made and the condition is one of posterior rupture, then the repair of the urethra is almost impossible because of the backward displacement of the prostatic portion. In a number of cases also the bleeding from the urethra is so free that it would seem necessary actively to deal with it and to incise the perineum, and this is, of course, imperative if extravasation has occurred.

2. Suprapubic cystostomy and splinting of the urethra by indwelling catheter without incising the perineum, a curved metal catheter being passed down the posterior urethra through the internal meatus and another through the external meatus along the anterior urethra, and the catheters gently manipulated until the beaks come in contact. The second catheter is then passed into the posterior urethra as the first is withdrawn, following its track. A rubber catheter can then be attached to the beak and by withdrawing the penile catheter the former is made to lie along the whole length of the urethra.

The indwelling catheter in this method does not drain urine, only acting as a splint. It would seem that if urine passes along the catheter the risk of urethritis is much greater.

The disadvantages here lie in the manipulations necessary to traverse the rupture, which may cause increased damage; the possible difficulty of traversing the rupture, greater than in posterior when the rupture can be felt if not seen; and the possible introduction of organisms along the urethra into the perineal hæmatoma, which is not drained as is the case in rupture of the posterior urethra where the suprapubic incision allows of the evacuation of the clot. If sepsis does occur it may lead to loss of a considerable length of urethra.

3. Suprapubic cystostomy and repair of the urethra at the same time. The actual damaged urethra may be dealt with in one of the following ways.

a. If is impossible to approximate the two ends of the urethra—the proximal end can always be found with ease by retrograde catheterization from the suprapubic wound—a catheter is threaded along the length of the urethra to act as a splint, the perineal wound being packed. The catheter tip should be short of the bladder so that no urine drains along the catheter. This treatment corresponds to the second method except that manipulations are carried out under vision and therefore produce less added trauma, hæmorrhage is dealt with, and the hæmatoma which might act as a nidus for infection is evacuated.

It is probably inadvisable in the presence of this acute trauma to attempt to mobilize the anterior urethra so as to allow of suture.

b. The roof of the urethra only is sutured and the perineal wound packed. No catheter need be left in. Suprapubic drainage is maintained, the patient being nursed in the Trendelenburg position. This method is considered by many to be the best way of dealing with the rupture and with the least likelihood of being followed by stricture formation.

One of the cases was dealt with in this way and was followed by a definite stricture. It should be noted that it was the only case of the three in which the catheter drained urine from the bladder and which was associated with definite urethral infection. It was also the one with most severe local trauma—this was noted by the local damage at the time of the operation, and is evidenced by the associated fracture of the ischiopubic ramus.

c. The whole of the urethra is sutured, the perineal wound being packed.

Suture of the urethra completely is said to be followed by severe stricture formation (Hamilton Bailey, 1928), but this I think is much less likely to follow if the suturing is not continuous and if the perineal wound is left open, so that no undue constriction of the urethra will take place, Nature carrying out the repair. The case in this small series dealt with in this way was healed and passing urine in three weeks' time and never showed any sign of stricture formation.

The suture of the urethra over a catheter, with closure of the perineal wound in layers, the catheter being removed at the end of the operation, is not advocated in the presence of acute trauma. The closure in layers is usually impossible, as hæmostasis is imperfect and constriction with stricture formation made more certain.

CONCLUSIONS

It is impossible to dogmatize from the treatment of three cases, but certain impressions have been gained.

a. Suprapubic Cystostomy: Many surgeons still deal with a complete rupture of the anterior urethra by a perineal operation only. There is no doubt that a preliminary suprapubic cystostomy renders the finding of the proximal end of the urethra a simple matter. Without it the prolonged search carried out adds needless local trauma and may be unsuccessful and necessitate ultimate cystostomy. Having performed the cystostomy, the deviation of the urine in this way would seem to be preferable in theory to drainage through the perineum or along the urethra. However, Banks (1927), who treated 3 cases of posterior rupture, closed the suprapubic wound and drained by indwelling catheter with good results.

b. Indwelling Urethral Catheter: Except when suture is impossible, it is not necessary to leave a catheter as a splint. It seems, however, less likely that infection of the urethra will occur if the urine does not drain along the catheter, and surgeons lacking in confidence can leave an indwelling catheter. It must be noted that where urine drains along the catheter urethritis is inevitable and stricture formation likely.

c. Details of Local Treatment of Urethra: Because no individual surgeon deals with any large number of cases, because in any series treatment carried out by the individual surgeon has differed in detail in his own personal cases, because in any review no mention has been made of the severity of the local trauma—and this is, in my view, an important factor in the production of stricture—it is impossible to lay down with confidence a method of treatment which will be followed with minimal or no stricture formation. In the cases recorded here, there was a great deal of local damage and urethral bruising in addition to the rupture in the third case, whilst in the second case the rupture was clean cut and the tissues in the neighbourhood had hardly been involved.

It is suggested that ruptures of the urethra should be dealt with at the time of the injury. To postpone this till a later date would be to subject these cases to a second operation, perineal section with excision of scar tissue and repair of the urethra, which could always be done on a stricture of the urethra if it had formed as a result of the primary operation. In the majority of cases, however, it is contended that either no stricture would occur following immediate perineal section, or were one to arise, it would

lend itself to dilatation. It is further suggested that a suprapubic cystostomy which helps with the localization of the proximal end of the urethra, should remain for the purpose of deviation of the urinary stream.

SUMMARY

1. Three cases of complete traumatic rupture of the urethra are described.

2. It is suggested that ruptures of the anterior urethra should be treated by suprapubic cystostomy, perineal section with suture of the roof, and packing of the perineal wound, urine draining only through the suprapubic wound.

3. Ruptures of the posterior urethra should have a suprapubic cystostomy, together with splinting of the rupture by an indwelling catheter.

4. The incidence of strictures following rupture of the urethra is increased by local sepsis. This may possibly be minimized by chemotherapy. The severity of the local damage is also a factor in its production.

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EXPERIMENTAL SURGERY

ARTERIAL SPASM

AN EXPERIMENTAL STUDY

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THE purpose of this paper is to report the experimental production of arterial spasm in the limbs of rabbits and to discuss the possible significance of our findings. We began this work in an attempt to find an explanation for some of the changes seen in the limbs of cases of 'crush syndrome'. Among the reports there were cases presenting clinical evidence of impairment of the circulation to the injured limb, e.g., coldness, œdema, and the development of blisters, but at autopsy there was no evidence of gross damage to the main arteries or of the presence of emboli. It occurred to us that the initial injury might have set up spasm in the main arteries, the spasm persisting after the release of the victim.

During the war of 1914–18 attention was drawn to cases where the main artery of a limb was found to be in spasm as a result of injury. Küttner and Baruch (1920) give a review of these cases, and among their own they saw all grades of ischæmia from coldness to incipient gangrene of the extremities where bullets or shrapnel had passed close to the main artery of a limb. The artery itself was not grossly damaged, though in many cases the accompanying vein and nerves had been severed. In their experience the condition tended to pass off within 18 to 36 hours of the injury, leaving no permanent ill effects; furthermore, cases left alone did just as well as those where the artery was explored and (usually) freed from the surrounding fascia.

Montgomery and Ireland (1935) give a further summary of recorded cases of segmental arterial spasm following injury, and add two cases of their own where the brachial artery was found to be in spasm a few hours after a supracondylar fracture in one case and an operation on the elbow in the other. They explored the artery because of signs of a severe ischæmia in the forearm. They exposed the artery and packed the wound with warm saline dressings, and the condition was relieved in a few hours.

It has long been believed by Leriche (1933) that Volkmann's ischæmic contracture is due to a spasmodic contraction of the arteries of the injured limb. One of us reported a case a few years ago that supports this idea. A man was admitted with a fracture of the lower end of the humerus, and when reduction was attempted it was found that the correct alinement of the fragments could only be maintained with the forearm in full extension. Accordingly the arm was enclosed in a plaster cast in this position, and the completion of the cast left to the nursing staff. A few moments after the cast had been finished an assistant surgeon walked into the room, and, believing that a mistake had been made in the application of the cast in extension, proceeded

to flex the elbow before the plaster had set. Twelve hours later the patient was complaining of severe pain in the hand and arm. The plaster was removed, and the limb found to be swollen and cyanotic, and there was complete paralysis and anæsthesia of the forearm and hand. As the circulation did not return after the removal of the plaster the brachial artery was explored under a general anæsthetic. The artery was found to be in spasm and to be reduced to about one-third of its normal size. An extensive peri-arterial sympathectomy was performed by stripping the vessel for the whole length of the arm. The circulation rapidly returned and a complete recovery of function took place within a few weeks (Trueta, 1934). The view that arterial spasm plays an important part in the production of Volkmann's ischæmic contracture is upheld by Griffiths (1940) in a recent review of the subject. Other cases have since been described where the brachial artery has been found to be in spasm in cases presenting all the early signs of Volkmann's contracture (Plewes, 1940; Seddon, 1941).

Occlusion of the main artery by itself is not enough to explain the severity of the ischæmic changes sometimes seen in these conditions, and the important factor undoubtedly is the state of the collateral circulation. This is not dealt with by Küttner and Baruch (1920) or by Montgomery and Ireland (1935), but it has been emphasized by Leriche and Stricker (1933) and again recently by Gage and Ochsner (1940). Apparently in certain cases when the artery is occluded either by spasm following injury, or by a surgical ligature, or by embolism, the whole collateral circulation is also thrown into a state of spasm. The local spasm of the damaged artery may be myogenic in origin, as suggested by Küttner and Baruch (1920), and this view is supported by the old observation that isolated strips of artery can be made to undergo a spastic contraction that will last for several days *in vitro* (MacWilliam, 1902). The spasm of the collateral vessels is probably reflex in origin, and Leriche believes that impulses ascending from the damaged segment stimulate the whole sympathetic nerve-supply to the affected limb, and possibly even that of the opposite limb. This theory remains unproven because of the failure to find the afferent nerves lying in the vessel walls (Leriche and Fontaine, 1928). A finding in accord with the views of Leriche is that excision of the injured segment of the injured artery will promptly relieve the spasm of the collateral circulation in some cases. The importance of the collateral circulation in cases of injury to the main artery has been stressed by Gage and Ochsner (1940). They state that the spasm of the collateral vessels will be completely relieved by the removal of the sympathetic nerve-supply to the affected limb. They add that no operation upon a main limb artery should be undertaken without first taking steps to paralyse the sympathetic supply to that limb.

Before describing our work we would like to draw attention to a case reported briefly by Griffiths (1940). A tourniquet (nature unspecified) was accidentally left in place for six hours; eighteen hours later, the symptoms of ischæmia persisting, the limb was explored and the femoral artery found to be in a state of spasm. In this connexion it is interesting to read that one of the reasons given by Esmarch (1877) for the introduction of the soft rubber tourniquet was that cases of gangrene sometimes followed the use of the inelastic tourniquet.

We therefore decided to see whether the application of an inelastic tourniquet could produce damage to the arterial supply of a limb that would persist after the release of the tourniquet.

EXPERIMENTAL INVESTIGATION

Brown rabbits were used, and the tourniquet consisted of soft wire placed inside a piece of rubber tubing to avoid injury to the skin. The wire was tightened with pliers until it was certain that the arterial blood-supply had been shut off. The legs were previously clipped, and in order to prevent the tourniquet slipping down the leg the wire was hooked over the ischial tuberosity before tightening; by this means pressure was exerted over the femoral vessels as they lay relatively exposed in the groin. The tourniquet was always put round the right leg, the left serving as a control. The tourniquets were left in place for 4½ hours; this time was chosen arbitrarily, and, as the preliminary results were satisfactory, was adhered to throughout the work. The tourniquet was applied after anæsthesia with intraperitoneal nembutal supplemented with ether; the animals remained drowsy while the tourniquet was in place, and showed no signs of discomfort. Arteriographs were taken of both limbs simultaneously at various intervals after the release of the tourniquet. For this purpose further doses of nembutal and ether were given. The abdomen was opened, and 5 c.c. of a 1–1 solution of sodium iodide in saline was injected into the aorta through a fine needle, the injection taking from 6 to 10 seconds. In all cases the anæsthesia had to be deep, for in lightly anæsthetized animals the injection of sodium iodide tended to be followed by violent muscular contractions of the limbs which spoiled the radiographs. A first exposure was made towards the end of the injection when 4½ c.c. had been given, and a second about 10 seconds later.*

Results.—In 5 normal animals arteriographs were taken under the same conditions of anæsthesia. The whole arterial and venous tree was seen to be well filled in the first arteriograph, the prominence of the veins being particularly impressive (*Fig.* 77).

* The hind limbs were held out over a wide-angled V-shaped support, a separate film being placed under each leg. Ilfex (non-screen) films were used with the following factors: tube-film distance 33 in., 63 kv., 80 ma., 0·2 sec.

In 4 animals the arteriographs were taken within an hour of the release of the tourniquet. In every case the artery of the right leg was contracted; sometimes the spasm was localized, but more often it appeared to affect the whole arterial tree, and the distal branches were either empty or else very poorly filled. In two of the animals there was, in addition, a well-marked spasm of the artery of the uninjured limb, the spasm beginning at a level corresponding with the site of the tourniquet on the other side. The veins were never visible in the first radiographs (*Fig.* 78), and were only faintly seen in some of the later ones.

altogether satisfactory, and the solution tended to diffuse away very rapidly. Arteriographs were taken after periods ranging from 15 to 40 minutes. Although a rather high proportion of the injections in these animals was faulty, the vascular picture was identical with that in the untreated animals.

In 5 other animals we removed the sympathetic chains lying alongside the aorta an hour after removing the tourniquets, and arteriographs were taken 24 to 48 hours later. In two of these animals where the injection was satisfactory the arteries showed a trace of spasm, but it was never so marked as in the untreated animals examined

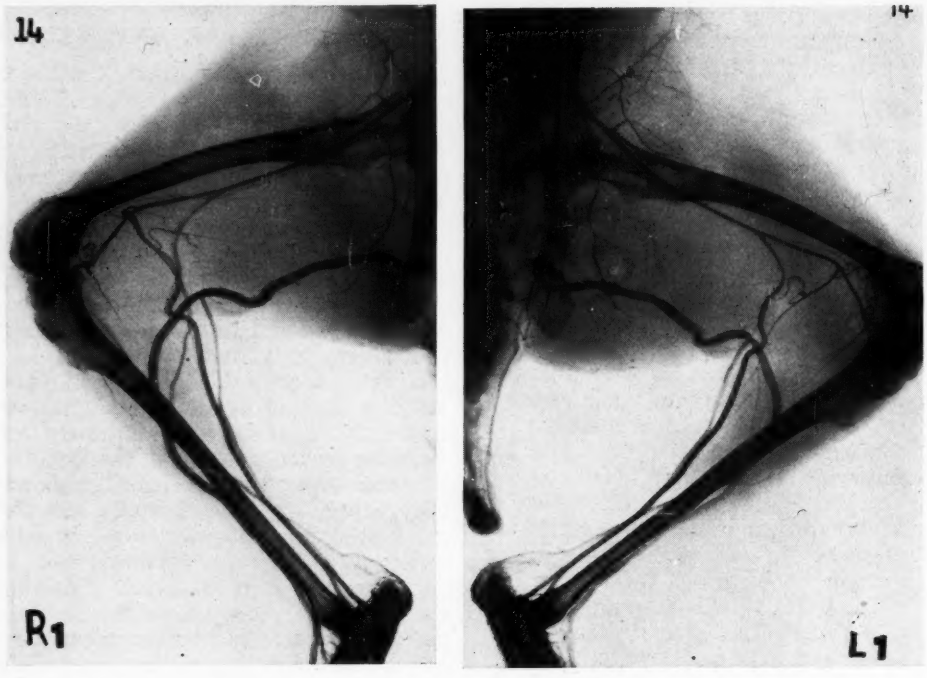

FIG. 77.—Arteriograph of normal rabbit. Note the complete filling of the vascular tree down to the heel. The veins are prominent.

In 9 other animals radiographs were taken at intervals of 24, 48, and 72 hours after the release of the tourniquet. In the two animals examined 72 hours after the release of the tourniquet where the injection was a good one the arterial spasm is seen to persist, and there is very poor filling of the arterial tree in both legs. In one animal of this group the circulation appeared to be normal, but in this case the limb had never become œdematous or paralysed, indicating that the tourniquet had not been adequately tightened.

Attempts to Relieve the Spasm.—We next endeavoured to find a means of relieving this severe and persistent spasm. We applied tourniquets to a further 6 animals, and about 30 minutes after their release the abdomen was opened and the tissues round the aorta and sympathetic chains infiltrated with 2 to 4 c.c. of a 2 per cent solution of novocain. Owing to the laxity of the tissues the infiltration was not

at this interval after the release of the tourniquet. Careful examination of the films suggested that a large number of the small vessels were filled, and this gives rise to a general increase of density in all the films. When the limbs were examined they were warm and the skin was red, and when incised before the death of the animal they bled profusely in contrast to all the other animals examined. The accompanying table summarizes the results we obtained. From this table it will be seen that a number of the injections are described as partial. All the injections of sodium iodide were given with the same needle and syringe and by the same person; yet, while no difficulty was experienced when injecting the normal animals, in quite a number of the experimental animals the needle apparently pierced the distal wall of the aorta towards the end of the injection. Although we have no actual proof that the aorta was diminished in size in some of these

animals, our experience with these injections suggests that this may possibly have been the case.

Effects upon the Muscles.—Though primarily interested in the behaviour of the vessels, we examined the legs after the death of the animal and removed pieces of muscle for section. We always took muscle from the thigh below the level at which the tourniquet had been applied, and also from the anterior tibial group of muscles. After removal of the tourniquet the leg rapidly became very œdematous, and within two hours of the removal gross œdema of the muscles and connective tissue was obvious to the naked eye. Sometimes the fluid was clear; at other times it was stained with blood. We never found any reason for these differences, but they may possibly have been due to differences in the tension of the tourniquet. This was a factor we were unable to control. Microscopically the muscle-fibres were broken up and separated from one another

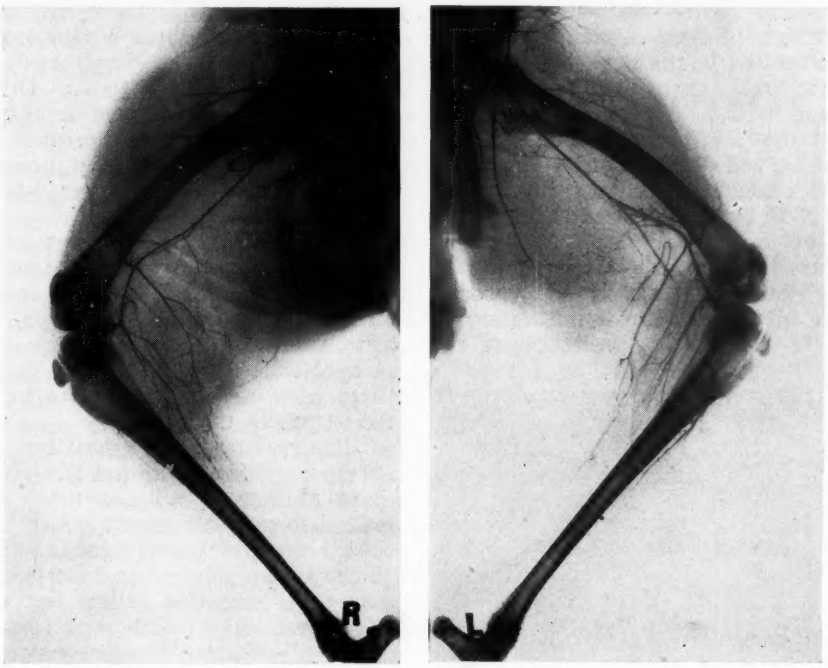

FIG. 78.—Arteriograph of rabbit one hour after the release of tourniquet. Note spasm of right femoral artery and the poor filling of its branches. Note also the spasm of the left femoral artery, commencing at the level corresponding to the site of the tourniquet on the other leg.

EXPERIMENTAL INVESTIGATION OF ARTERIAL SPASM: SUMMARY OF RESULTS

PROCEDURE	INTERVAL BETWEEN RELEASE AND X-RAY	NUMBER OF ANIMALS	NUMBER OF PARTIAL INJECTIONS*	RESULTS
Normal rabbits	—	5	0	Arterial and venous filling complete in all
Tourniquet in place for 4½ hr.	½–1 hr.	4	1	Spasm on right side in 4; collateral spasm in 2
Tourniquet in place for 4½ hr.	24 hr.	3	0	Spasm on both sides in 2; leg normal in 1
Tourniquet in place for 4½ hr.	48 hr.	3	2	Spasm well marked in 1
Tourniquet in place for 4½ hr.	72 hr.	3	1	Bilateral spasm in 2
Tourniquet in place for 4½ hr.	14 dys.	1	0	Vessels as in normal animal; muscles showed Volkmann's contracture
Tourniquet for 4½ hr.: Infiltration with *Novocain*	1–2 hr.	6	4	Spasm well marked in 3 animals
Tourniquet for 4½ hrs.: *Sympathectomy* ½ hr. after release of wire	24 hr. (3) 48 hr. (2)	5	2	Spasm persisting in 1; denser leg shadows suggesting filling of collaterals in 4

* In these animals all the sodium iodide did not enter the aorta, and in some the radiographs showed little or no filling of any of the main vessels. In others the filling was incomplete, but spasm of the upper parts was clearly visible.

by the exudate. After 24 hours a cellular infiltration of polymorphonuclear leucocytes was apparent, and this was later followed by mononuclear cells. Sections of muscles taken 72 hours after the release of the tourniquet showed groups of fibres that were dying, and there was an extensive infiltration of large mononuclear cells.

We were interested in the persistent stiffness of some of these limbs, and allowed two rabbits to live for 14 and 21 days after the release of the tourniquet. In these animals some of the muscles of the thigh and leg were yellowish green in colour and of firm consistency. Sections showed the typical picture of Volkmann's contracture. There were islands of completely necrotic fibres—muscle sequestra (Griffiths)—surrounded by fibrous tissue, and other areas of partially damaged fibres with an extensive infiltration of fibrous tissue. *Fig.* 79 shows a section of the

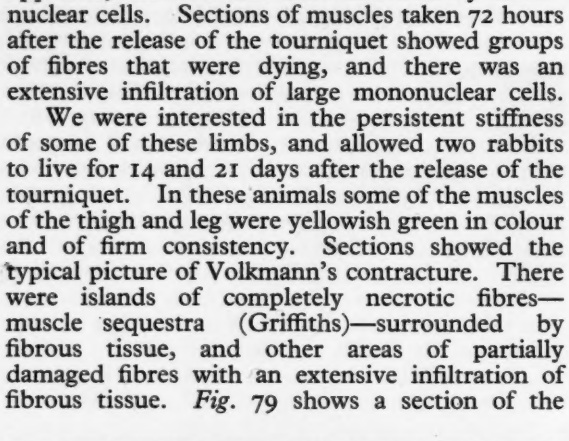

FIG. 79.—Section of the tibialis anterior muscle from a rabbit 14 days after the release of tourniquet.

tibialis anterior muscle from a rabbit killed 14 days after the release of the tourniquet. Gangrene of the extremities was not seen in any of these animals, though the legs felt cold for a few days after the release of the tourniquet. In no case did we find any evidence of thrombosis of the main arteries, but in one case ante-mortem thrombosis of the veins had taken place.

DISCUSSION

It is dangerous to draw conclusions from animal experiments and apply them directly to man. We do feel justified, however, in emphasizing three points that arise out of this work. They are the method of producing the spasm, the severity and persistence of the ischæmia, and finally the appearance of arterial spasm in the opposite and uninjured limb.

That an artery responds to direct violence by going into spasm is a fact well known to operating surgeons. This direct type of injury will explain the spasm seen in cases during the last war when missiles passed close to the main arteries; it will also explain those cases of Volkmann's contracture following a fracture where a bony spicule has actually damaged the vessel wall. What is not so well known is that a less direct type of injury may give rise to a similar picture. Thus Volkmann's contracture may follow a crush or blow that does not fracture the bones (Barnard, 1901; Dudgeon, 1902; Willis, 1901). In the case described by Trueta, and referred to earlier, the cause of the arterial spasm seems to have been pressure from the flexed plaster. In the same way a tourniquet may give rise to a persistent arterial spasm (Griffiths, 1940). We do not suggest that our tourniquet bears any close resemblance to any that might be used in ordinary medical practice; on the other hand, a similar type of injury might conceivably be produced by the continued pressure of fallen masonry, particularly should the sharp edge of a beam or brick be pressed on to the vessels in the groin.

The reasons for the persistence of the spasm are more difficult to assess. In cases where spasm followed bullet wounds the condition tended to pass off spontaneously and apparently caused no permanent damage to the structures of the limb (Küttner and Baruch, 1920). This may have been due to the fact that the initial injury was either mild or transient. In certain cases of Volkmann's contracture a bony spicule may be lodged against the artery wall and give rise to a persistent irritation. This will not explain those cases where no fracture has occurred. In our experiments the tourniquet was always left in place for the same length of time and yet in the majority of animals the spasm persisted. We have remarked earlier on the differences that were seen in the condition of the limbs when examined just before death, and we believe that this may have been due to a difference in the tension of the tourniquet. The importance of the type of injury was borne out by our experiences in the use of a different type of tourniquet. Another series of rabbits used for different experiments had soft rubber tourniquets applied for the same length of time. Though the legs became swollen after their release the persistent stiffness and paralysis were never noticed, and arteriographs done on two of these animals showed a normal vascular picture. This may explain the failure of Griffiths to produce Volkmann's contracture in the limbs of rabbits by means of tourniquets.

The pronounced effect this arterial spasm may have upon the blood-supply to the leg is illustrated by the complete absence of venous shadows in the radiographs in our tourniquet experiments. In those animals in which arteriographs were taken within a few hours of the removal of the tourniquet this apparent sluggishness of the circulation might be explained by the condition of 'shock' that may follow the release of the tourniquet from the legs. Such animals do

get a hæmoconcentration, but the blood-picture returns to normal within 24 hours. This explanation will not hold, therefore, for those animals presenting the same circulatory picture 72 hours after the release of the tourniquet. The fact that a condition indistinguishable from Volkmann's ischæmic contracture will develop in some of these animals is sufficient indication of the severity of the local anæmia. In this connexion it is interesting to note that Griffiths (1940) was able to produce such a change in the muscles of the rabbit only by tying the common iliac and common femoral arteries on the same side. It is clear that not only the main artery is affected, but also the whole of the collateral circulation as well.

The third and most interesting feature of these experiments was the appearance of a symmetrical spasm in the main artery of the uninjured limb. Traces of the spasm and evidence of a defective circulation were seen in arteriographs taken 72 hours after the release of the tourniquet; the resultant ischæmia was never sufficiently severe to interfere grossly with the function of the limb, and it did not become œdematous. The significance of this spasm lies in the apparently widespread vascular reaction to a comparatively localized trauma. In the case of the leg no ill effects are noticeable,* but this might not necessarily be the case were the vessels serving a more vital organ injured and sent into spasm.

Regarding the effects of the injection of novocain and of sympathectomy, we wish to say little. The failure of the novocain to release the spasm in our animals may have been due to the impossibility of getting a satisfactory infiltration round the nerves on account of the laxity of the rabbit's retroperitoneal tissues, or it may have been due to the weakness of the solution used. On the other hand, our experience does fit in with a very interesting clinical observation made by Seddon (1941). An early case of Volkmann's contracture was seen in a patient with a supracondylar fracture with gross ischæmia of the limb. A brachial plexus block was carried out which must have affected the sympathetic fibres, for the patient developed a well-marked Horner's syndrome. The signs of ischæmia persisted unaltered, and $3\frac{3}{4}$ hours later the arm was explored under a general anæsthetic and the brachial artery found to be in spasm. Resection of the injured segment was followed by a prompt restoration of the circulation. In the animals where a sympathectomy was performed there was a marked improvement in the circulation to the limb even though traces of spasm were still to be seen in the main artery. The legs were red and warm, and when incised just before the animal was killed the muscles bled profusely. It would seem that while the local effect on the damaged artery may be myogenic in origin, the reflex closing of the collateral circulation is brought about by the stimulation of the sympathetics.

* Since this was written an interesting case has been reported by Atlas (*Surg., Gynecol. and Obst.*, 1942, **74**, 236). A small embolus became lodged in the tibial artery of one leg, and gave rise to such extensive reflex vasospasm involving both legs that it was thought at first that the obstruction had occurred at the bifurcation of the aorta.

SUMMARY

1. A severe and persistent arterial spasm can be produced in the legs of rabbits by means of a tight wire tourniquet left in place for $4\frac{1}{2}$ hours.

2. The spasm has been seen to persist for as long as 72 hours, and in animals allowed to survive for longer a condition of the muscles similar to Volkmann's ischæmic contracture develops.

3. The spasm affects the main arteries and also the collaterals of the injured limb. Segmental spasm of the main artery of the opposite uninjured limb is also clearly seen in many of the animals.

4. Local infiltration of the sympathetics with novocain failed to relieve the spasm within 40 minutes. Removal of the sympathetic chain after release of the tourniquet improved the general circulation to the limb, although traces of spasm in the main trunks were often seen.

5. The relation between these experimental findings and certain clinical conditions of ischæmia are discussed.

We wish to express our gratitude to the Director of the Nuffield Institute of Medical Research for permission to carry out part of this research at the Institute, and to Miss M. M. L. Pritchard and Mr. M. S. Tuckey for their help in the arteriography. We should also like to thank Dr. A. E. Barclay for his interest and advice throughout the work. Dr. A. H. T. Robb-Smith kindly examined the sections for us.

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SHORT NOTES ON RARE OR OBSCURE CASES

ACUTE PHLEGMONOUS ENTERITIS

By T. G. I. JAMES, London

Acute infection of the wall of the small intestine by pyogenic micro-organisms is a condition which occasionally enters the arena of surgical emergencies. The successful outcome of the following case is the reason for its presentation.

CASE REPORT

History.—W. H., a male aged 64, was admitted to hospital with severe abdominal pain. Three days before, he had sustained a fall, but apart from an abrasion of the right elbow and some vague pain in the back, he had suffered no other injury. The day before admission he was seized with acute generalized abdominal pain, which later became localized to the right of the umbilicus. He had no nausea or vomiting. His previous health had not been disturbed since an occasion twenty years before, when he had undergone an operation for the cure of a right inguinal hernia.

Fig. 80.—External appearance of the gut.

On Examination.—The patient looked ill. The temperature was 99·6° F., the pulse-rate 76, and the respirations 22. The abdomen was not distended, but showed moderate tenderness and rigidity all over, most marked in the right lower quadrant. There was no tenderness over the back, and apart from some superficial abrasions over the right elbow there were no other abnormal findings. A diagnosis of appendicitis was made and he was submitted to operation.

At Operation.—The cæcum was found fixed to the scar of his hernia operation, but there was no trace of his appendix (it subsequently transpired that this had been removed at the time of the herniotomy). The stomach, gall-bladder, duodenum, and colon were normal. In the left upper quadrant a mass was felt about the size of a small sausage. This was drawn into the wound and was found to be in the jejunum about 12 in. from the duodeno-jejunal junction. After wiping away the enveloping omentum, about 3 in. of the gut was found rigid and thickened to about twice its normal size. The peritoneal coat, flaked lightly with lymph, was acutely inflamed, and on the anterior surface there were two small yellow areas, which gave the impression of abscesses about to perforate (*Fig.* 80). The neighbouring mesentery was inflamed, but contained no abscesses or enlarged lymph-glands. There was no free fluid in the peritoneal cavity. The affected part of the gut was removed and an end-to-end anastomosis made. Convalescence was uninterrupted and recovery complete.

The Specimen.—The resected portion was opened (*Fig.* 81). The jejunal folds were large and œdematous—particularly the submucosa. The mucosal surface was acutely inflamed, and in three of the mucosal folds there were abscesses, two of these extending to the peritoneal surface. A small quantity of pus was found in each abscess and cultures of these revealed *B. coli*. There was no ulceration of the mucosa and no evidence of a foreign body. The pathologist's report on the histological section was: "Acute phlegmonous inflammation of the small intestine. The œdema and infiltration with inflammatory cells—chiefly polymorphonuclears—is present throughout the whole width of the gut".

COMMENTARY

Acute phlegmonous inflammation of the small intestine is much less common than in the stomach. Rokitansky[1] drew attention to the condition in 1842, and since then several cases have been reported. Clark and Wright[2] in 1937 gave a very complete review of the subject—reviewing 42 cases in the literature, including 2 of their own. According to their analysis the jejunum is more frequently involved than any part of the small intestine. The condition is

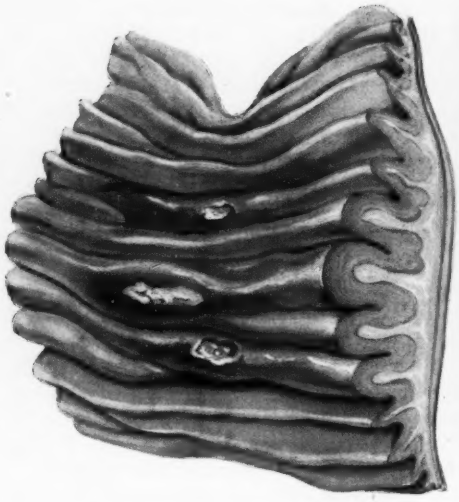

Fig. 81.—Internal aspect of the gut.

more common in males, and the sixth decade claims the majority of cases. A wide variety of organisms has been held to be responsible for the condition—streptococci being more frequently encountered than staphylococci, pneumococci, or *B. coli*. Although defects in the mucosa have rarely been seen, it would appear that the infection is of enterogenous origin. The association in some of the reported cases of penetration of the mucosa by sharp foreign bodies lends support to this assumption. A few cases have been attributed to abdominal trauma, although subsequent examination has revealed no defect in the mucosa. The length of intestine involved may be as little as 4 cm., or as much as 60 cm. As in the case here reported, areas of suppuration are common and may penetrate the mucosa, producing ulceration, but more frequently they extend to the peritoneal surface, causing local or generalized peritonitis. The neighbouring mesentery is often œdematous and may show areas of suppuration, with or without similar involvement of the mesenteric glands.

When the peritoneal surface is involved symptoms and signs of local or general peritonitis appear. The clinical picture simulates acute appendicitis and it is not possible—unless, as occasionally happens, a mass is present—to make a pre-operative diagnosis.

Of the 42 cases analysed by Clark and Wright, 35 died—22 without surgical intervention. Only 10 were subjected to resection, and of these 7 recovered. Resection of the involved segment of the gut is thus the treatment of choice.

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A CASE OF ADAMANTINOMA OF THE FEMUR

BY A. LINCOLN BELL
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ADAMANTINOMA or epithelial odontome of the jaw is of comparatively frequent occurrence among natives of Nigeria, but this is the first case to be recorded in which the tumour occurred in the femur.

CASE REPORT

HISTORY.—The patient, an African boy aged 16, presented himself at the out-patient department of the African Hospital, Victoria, Cameroons, complaining of pain in the left leg and knee, accompanied by swelling of the lower end of the femur.

The pain was increased on movement. He stated that the condition had been present for about six weeks and that it was getting worse.

ON EXAMINATION.—A diffuse firm swelling of the left knee and lower part of the femur was found. The swelling did not fluctuate and was immobile. The knee was in full extension and any attempt at flexion was productive of a moderate degree of pain. There was a small effusion into the joint.

The case was regarded as one of deep intramuscular abscess. This condition is common in the Cameroons and some swellings reach huge dimensions.

The patient was admitted and his temperature was found to vary between 98° and 100° F.

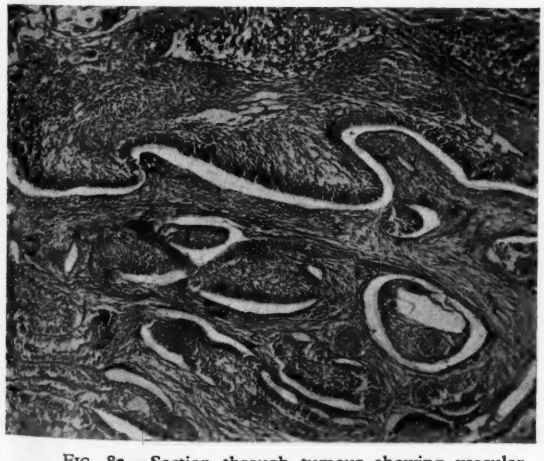

FIG. 82.—Section through tumour showing vascular fibrous stroma in which are embedded islands and anastomosing strands of cellular tissue. (× 40.)

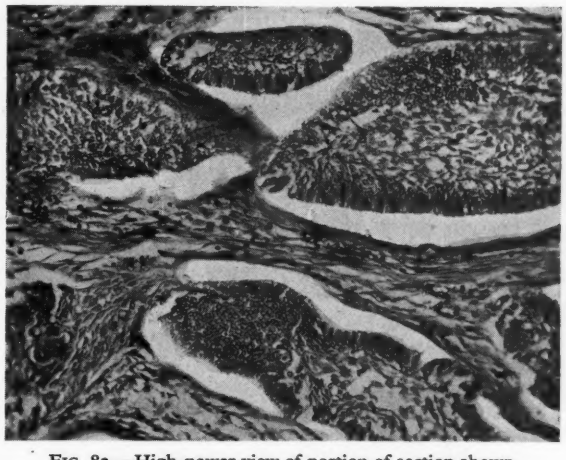

FIG. 83.—High-power view of portion of section shown in *Fig.* 82, showing areas composed of a central mass of spindle-shaped or somewhat stellate cells with a surrounding peripheral layer of high columnar epithelium. (× 115.)

Two days after admission an exploratory excision was made for pus over the area of greatest swelling, a point 2 in. above the internal and upper border of the left patella. The incision opened into a fleshy growth which had not penetrated the deep fascia superficially, but which involved all the deeper tissues, including bone. The incision was extended down to the femur, when it was possible to palpate

numerous irregular bony spicules and masses in the deeper parts. These bony masses, though small, were larger than those met with in periosteal sarcomata. The femur was extensively involved.

SUBSEQUENT PROGRESS.—At the patient's request he was discharged from hospital, but he returned after five days and was re-admitted. His condition deteriorated rapidly and he died five weeks later with signs of a well-marked bronchopneumonia which had been present for three days before death. During these last weeks pain had been a marked feature, and it was always localized to the tumour area.

A post-mortem examination was not possible, but there was no clinical evidence to suggest the presence of secondary deposits.

HISTOLOGICAL REPORT (*Figs.* 82, 83).—A portion of the tumour, which was removed during the exploratory examination, was sent to the Medical Research Institute, Yaba. Sections were prepared and showed a vascular fibrous stroma in which were embedded islands and anastomosing strands of cellular tissue. Under higher power these areas were seen to be composed of a central mass of spindle-shaped or somewhat stellate cells with a surrounding peripheral layer of high columnar epithelium. In many places the central cell masses had undergone vacuolation and degeneration, resulting in the formation of cystic spaces. More rarely the cells of the central regions resemble squamous epithelium and an attempt at pearl or cell-nest formation is recognizable.

The structure in general is typical of a glandular adamantinoma,[1] or epithelial odontome.[2] Such tumours have frequently been recorded from the maxilla and mandible of Africans, and 13 cases have been reported (Smith and Elmes[3]). The present case, however, is the first instance in which such a tumour has been recorded from Nigeria in a long bone. Ewing[4] and Boyd[5] mention cases in which these tumours have occurred in the tibia.

SUMMARY

A case of adamantinoma of the femur, in an African, is reported, together with a description of the histological findings.

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PAPILLIFEROUS CARCINOMA IN A DOUBLE KIDNEY

BY RALPH SHACKMAN AND B. R. BILLIMORIA
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THE interesting features of the following case merit it being placed on record.

CASE REPORT

HISTORY.—C. C., aged 64 years, a labourer, was admitted to Hammersmith Hospital on July 7, 1939, with hæmaturia. For the previous seven months he had noticed intermittent attacks of hæmaturia, when the urine was intimately mixed with blood which was bright red in colour. The sequence of events during micturition was as follows: dirty urine—bright blood—clear urine. Occasionally small clots were passed. Hæmaturia was more marked after exercise. There was no dysuria and the frequency of micturition was D/N: hourly /1. He thought that he had lost a little weight recently. The past history presented no relevant features.

ON EXAMINATION.—A healthy-looking man of good build and colour. The cardiovascular, respiratory, and central nervous systems presented no clinical abnormalities. The blood-pressure was 150/100. The lower poles of both kidneys were just palpable, but no other abnormal signs were found on abdominal or rectal examination. The scrotum and its contents were normal.

Investigations.—Cystoscopy revealed two ureteric orifices on the left side, with blood coming from the lower orifice. A papilloma, ¼ in. in diameter, was seen just above the region of the double ureteric openings. The rest of the bladder appeared normal and a single normal right ureteric orifice had a clear efflux.

The urine contained numerous red blood-corpuscles and pus cells. The cultures were sterile. Blood-count: R. B. C., 4,200,000; W. B. C., 4000; and Hb, 84 per cent. Blood-urea: 40 mg. per cent.

A plain radiograph of the urinary tract showed no abnormalities.

Under low spinal anæsthesia, the vesical papilloma was fulgurated and an ascending pyelogram carried out (July 22).

The X-ray report (Dr. Duncan White) stated: "Two opaque catheters are present, demonstrating double left ureter. The opaque medium demonstrates a lower renal pelvis with normal calices and ureter. The upper renal pelvis and ureter show irregular mottled filling defects in the pelvis and the upper ureter. The calices cannot be distinguished, and the pelvis is dilated. The appearances suggest an extensive lesion in the upper pelvis and the upper ureter: I think, most probably, a neoplasm of the papillomatous type; however, it is not possible to exclude a chronic infective lesion" (*Fig.* 84).

Excretion urography proved the presence, function, and apparent normality of the right kidney.

OPERATION (July 24).—Under gas-oxygen and ether anæsthesia, an oblique incision was made in the left loin and a single left kidney was easily delivered. The upper pole was a little adherent to the perinephric fat and the organ was slightly larger than normal. It was divided transversely, by a line of demarcation, into an apparently normal lower half with a normal ureter and an intrarenal type of pelvis, and a cystic upper half with a dilated and firm pelvis (as if it contained 'growth'). Both ureters were mobilized down to the pelvic brim, where they were ligated and divided with a diathermy. Nephrectomy was completed and the wound was closed. The

patient was then placed in Trendelenburg's position and through a midline incision the lower ends of both left ureters were excised extraperitoneally, using a diathermy to divide them at the bladder. The wound was closed with drainage.

PROGRESS.—Convalescence was smooth and the patient was discharged home on Aug. 25 (32 days after operation). Adequate follow-up was interrupted by the war, but in September, 1941 (27 months after operation), the patient wrote to say that he was keeping fit and quite free from symptoms, and had gained 2 st. in weight since leaving the hospital.

FIG. 84.—Ascending pyelograms.

PATHOLOGICAL REPORT (Dr. S. Richardson).—"The tumour is a papilloma involving the pelvis and the ureter supplying the upper half of the kidney. It has produced obstruction with consequent hydronephrotic atrophy of the upper half of the kidney. The tumour is composed of numerous branching blood-vessels covered by several layers of epithelium. The nuclei are oval and arranged in a regular manner. Mitoses are not common. There is no invasion of surrounding structures. The ureter from the lower half of the kidney is patent and free from tumour. The lower half of the kidney shows no abnormality" (*Fig.* 85).

DISCUSSION

Duplication of the ureters is not uncommon. It is said to occur in about 4 per cent of cases (Lessig). The unilateral type of duplication is more common than the bilateral type. Usually the upper pelvis is smaller than the lower, but exceptions to this have been described, and, as in the case reported here, the upper pelvis may be larger than the lower one. However, this may have been associated with hydronephrotic atrophy of the upper half of the kidney.

The pyelogram shows, typically, how the upper ureter leaves the kidney on the medial side of the lower ureter and crosses behind the latter; in the lower third of its course it re-crosses the lower ureter and again lies medial to it. Cystoscopy confirmed the Weigart-Meyer 'law' that "the ureter of the upper half of a double organ opens caudal and medial to the ureter of the lower half" (MacAlpine). Of course, there must be exceptions to this rule.

Obstruction, inflammation, stone formation, and new growths may all occur in kidneys with duplicated ureters. In fact, there is evidence that such congenitally abnormal urinary systems are very susceptible to certain pathological changes. The case described in this article demonstrates two of these changes, namely, obstruction and new-growth formation.

The papilliferous type of tumour is the commonest one to be found in the renal pelvis, and its peculiar potential danger of spreading to involve the ureter and bladder is well recognized. In 46 collected cases, McConn found extension to the ureter in 23, and to the ureteric orifice and bladder in 17.

The cystoscopic finding of a small papilloma in the bladder, together with bleeding from a

ureter, suggested the possibility of this condition in our case. The irregular mottled defects of the ureter, seen in the pyelogram, were probably caused by multiple small blood-clots, because the specimen did not reveal any macroscopical evidence of ureteric implantation or spread.

As the lower half of the kidney and its associated ureter appeared normal, the possibility of partial nephrectomy with excision of the affected ureter was considered. But at operation, the natural line of separation between the portions of the kidney was indistinct. Furthermore, the risk of cutting through neoplastic tissue, together with the chance of leaving a urinary fistula, were so great that complete extirpation of the kidney and both ureters was carried out.

We wish to thank Dr. Allen Daley, Chief Medical Officer of the London County Council, and Professor G. Grey Turner for permission to publish this case.

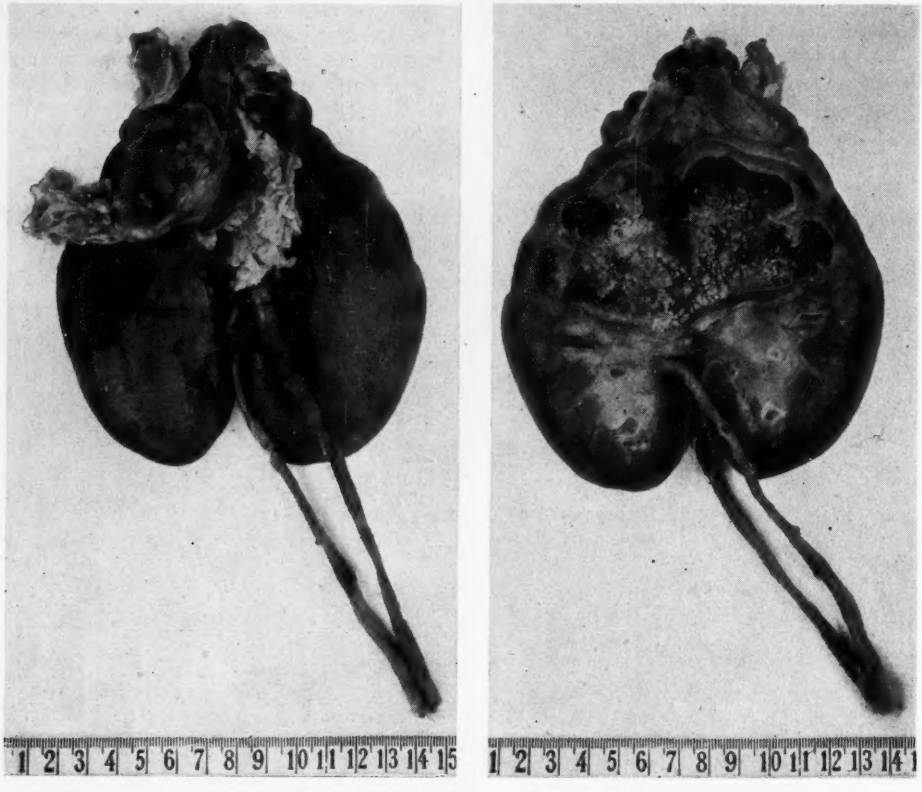

FIG. 85.—The left kidney and ureters after removal (*see text*).

Our thanks are also due to Dr. Duncan White and Dr. S. Richardson for the radiological and pathological reports.

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TRANSVERSE RUPTURE OF THE MESENTERY, COMPLICATED BY ARTERIAL EMBOLISM

BY BRYAN C. MURLESS
SURGEON LIEUTENANT COMMANDER, R.N.V.R.

A RUPTURE of the mesentery involving branches of the superior mesenteric artery is an unusual and serious abdominal injury. I can find few similar cases described in the literature. With the added interest of an arterial embolism occurring as a complication and the eventual recovery of the patient, the case seems worthy to be placed on record.

CASE REPORT

HISTORY.—A goods checker, aged 34, had his abdomen crushed between the buffers of two railway waggons. He was not at first very distressed and was driven to hospital in a van.

ON EXAMINATION.—Twenty minutes after the accident he appeared slightly shocked. Pulse-rate 72, and temperature 98° F. The abdominal wall and back showed no external evidence of trauma.

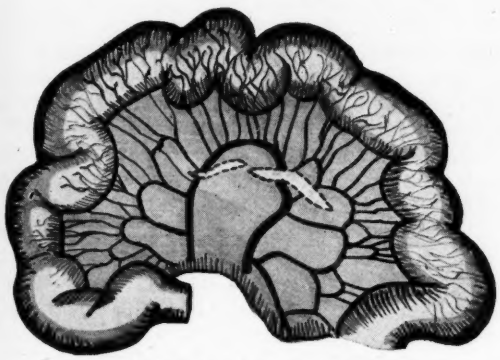

FIG. 86.—Sketch of injury to mesentery.

Palpation revealed some tenderness and slight rigidity around and below the umbilicus. Treatment for shock was instituted, but his general condition deteriorated rapidly. After thirty minutes, a rising pulse-rate, pallor, and increasing dullness in the flanks made the diagnosis of internal hæmorrhage unmistakable.

A pint of stored blood and ¼ gr. morphia caused some improvement in his condition immediately preceding operation.

A diagnosis of probable ruptured spleen was made. An exploratory operation was performed exactly an hour and a half after the accident occurred.

FIRST OPERATION.—Under chloroform and ether, followed by open ether, anæsthesia. The abdomen was opened by an upper midline incision and the peritoneal cavity found to be full of blood. The spleen and liver appeared undamaged. A rapid search revealed two transverse tears in the mesentery of the small intestine measuring 1 in. and 3 in. respectively and situated about 1½ in. from the mesenteric border of the gut (*Fig.* 86). The wall of the intestine was undamaged. A large branch of the superior mesenteric artery was spurting at the proximal edge of the longer tear. Bleeding was controlled and a further transfusion of two pints of stored blood started immediately.

The bowel was examined and a large loop at the lower end of the ileum, reaching to within 4 in. of the cæcum, had turned a blue-red colour. A rapid resection was performed. End-to-end anastomosis was chosen in preference to ileo-cæcostomy to save time, as the patient's general condition was not good. No attempt was made to swab out more blood from the peritoneal cavity. The abdomen was closed without drainage.

The resected small intestine measured 5 ft.

POST-OPERATIVE PROGRESS.—Immediate recovery from operative shock was rapid and well maintained. On the fourth day considerable distension and occasional vomiting caused anxiety. On the fifth day increasing vomiting necessitated continuous aspiration of the stomach contents. Four pints of intravenous glucose saline were given by a slow drip.

On the evening of the sixth day the patient complained of pain in the right foot, extending up the posterior surface of the right calf. The foot appeared normal and as the continuous saline had been given via a cannula in the right ankle vein, a phlebitis of this vein was suspected at first.

During the night flatus was passed for the first time since operation, followed by two natural actions of the bowel.

The pain in the right foot became increasingly severe and was not relieved by morphia. On the morning of the seventh day examination showed the right foot to be blanched and cold, with a bluish tinge at the tips of the toes. Sensation to touch was diminished. Pulsation could be felt in the right femoral artery, but was doubtful in the right popliteal. No pulsation could be felt in the anterior tibial artery on this side. Pain still persisted in the foot and the posterior surface of the leg.

The abdominal condition was now satisfactory, the distension having greatly decreased and the vomiting almost stopped.

A diagnosis of arterial embolism of the right popliteal artery was made. No abnormality could be discovered in the heart or lungs.

As the patient's general condition contra-indicated any extensive procedure and the time limit for successful embolectomy was probably passed, this operation was not attempted. A peri-arterial sympathectomy of the right femoral artery was performed by Sir William de Courcy Wheeler, Surgical Consultant to the Navy in Scotland, who was called in consultation.

FIG. 87.—Amputated foot, showing extent of gangrene.

SECOND OPERATION.—A quarter of an inch of the first part of the right femoral artery was stripped of its sympathetic fibres under local infiltration anæsthesia. The affected foot and lower leg were cleaned with spirit, wrapped in cotton-wool, and radiant heat applied.

EFFECTS OF OPERATION.—No immediate effect was observed from the operation. After six hours

the blanched appearance of the foot had improved, but the toes remained cold and the bluish tinge persisted. After twenty-four hours the big toe showed evidence of early gangrene, but the rest of the foot remained unchanged.

At this stage a small pulmonary embolism caused pain in the left side of the chest, with a slightly blood-stained sputum. These symptoms persisted for twenty-four hours.

During the next ten days a dry gangrene affecting the sole and anterior part of the dorsum of the foot developed very slowly. A definite line of demarcation was not complete till the fifteenth day after the second operation (*Fig.* 87).

The patient's general condition was now greatly improved and amputation was considered justifiable.

THIRD OPERATION.—Anæsthesia used: sodium pentothal followed by gas and oxygen. A rapid amputation through the leg was performed to leave a 6-in. stump, using anterior and posterior flaps.

It was not ascertained at operation whether the main vessels were patent, but on removal of the tourniquet general bleeding seemed to be less than usual.

POST-OPERATIVE HISTORY.—Healing of the flaps was slow and a small slough developed in the scar. When healing was complete an excellent stump resulted. Further recovery was uneventful.

The function of the intestinal tract was normal on discharge from hospital.

RADIOLOGICAL EXAMINATION.—Radiological examination of the intestinal tract was undertaken nine weeks after the original operation. A barium enema filled the large intestine normally and no stricture could be observed in the lower ileum. A barium meal filled the large intestine as far as the splenic flexure within three hours. There was no barium in the large intestine at one-and-a-half hours. No narrowing could be observed in the lower ileum.

DISCUSSION

Some interesting points arise from this case which are not fully discussed in the text-books of surgery.

As the incidence of this injury is rare it follows that the mortality-rates given by various writers show wide variation. Most figures include cases with tearing of the intestinal wall and cases of mesenteric damage without serious internal hæmorrhage. Massie[1] quotes a figure of 78 per cent in all subcutaneous injuries to intestines and mesentery. Sencert and Ferry[2] report 34 cases of various types of injury to mesentery and intestine, of which 13 recovered. The most likely explanation of a transverse tear in the mesentery is an injury which forces the small intestine as a mass away from its mesenteric attachment to the posterior abdominal wall. This can be well visualized in the above case where the abdomen received a glancing crush between two waggon buffers. A point of note is the absence of evidence of trauma on the skin of the abdominal wall or back. Totten[3] points out that in cases of injury to intestine or mesentery this is often the case, and quotes 25 cases in which only 8 showed external evidence of trauma. Delay in the onset of shock was remarkable in this case. Rupture of so large a mesenteric vessel might well be expected to produce collapse in a matter of minutes. It is noteworthy that the patient was driven to hospital sitting in a van, and that three-quarters of an hour had elapsed before shock became apparent.

Ileocæcostomy is the operation of choice after resections of ileum near the cæcum, but the time saved by end-to-end anastomosis is a consideration where severe hæmorrhage has rendered the patient desperately ill.

Sencert and Ferry quote a successful case where the ends of intestine were brought out and Paul's tubes used.

Bost,[4] after experiments on dogs, reports 3 successes in humans from suturing omentum around the affected loop of intestine with the object of supplementing the failing blood-supply; the mesenteric tears were $3\frac{1}{2}$ to 4 in. long and the blood-supply to the gut was "apparently cut off", but he does not mention any colour changes. However, the standard teaching of resection and anastomosis, although carrying a very high mortality-rate, seems to offer the best chance of recovery.

An embolism in the right popliteal artery on the evening of the sixth day was a distressing occurrence and must be a very rare post-operative complication in cases without cardiac disease. The pathology of such an embolism seems somewhat obscure, but a possible explanation in the case under discussion might be the dislodgement of a clot from the abdominal aorta, the intima of which had been damaged by the original crushing injury, or by a pull on the superior mesenteric artery and its mesentery.

In different circumstances embolectomy might have been successfully undertaken. Sympathectomy of the femoral artery seemed the second best choice. It was hoped that a transient vascular dilatation following the operation might limit the speed of the gangrene, and promote the formation of a line of demarcation in the region of the foot.

Amputation was delayed as long as possible to ensure recovery from the effects of the abdominal operation. When finally undertaken it was well tolerated and followed by rapid improvement in the general condition.

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[2] SENCERT, L., and FERRY, G., *Jour. de Chir.*, 1921, **18**, 561.
[3] TOTTEN, H. P., *Surgery*, 1938, **4**, 597.
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REVIEWS AND NOTICES OF BOOKS

Acute Injuries of the Head. Their Diagnosis, Treatment, Complications, and Sequels. By G. F. ROWBOTHAM, B.Sc. (Manch.), F.R.C.S. (Eng.), Late Hunterian Professor of Surgery and Dickinson Scholar; Surgeon in Charge, Department of Neurological Surgery, Newcastle General Hospital; Neurological Surgeon, Royal Victoria Infirmary, Newcastle-on-Tyne; etc. With a Foreword by NORMAN M. DOTT, M.B., Ch.B. (Ed.), F.R.C.S. (Ed.). $9\frac{3}{4} \times 6\frac{3}{4}$ in. Pp. 288 + xii, with 124 illustrations. 1942. Edinburgh: E. & S. Livingstone. 25s. net.

No matter how acute paper shortage may be, there can be no adverse criticism of the appearance of a book of this kind at the present time. Head injuries constitute a subject on which guidance is much needed, particularly in war-time, and the author of this book has produced a clearly written and attractive account of the subject, which should have a wide circulation because of its wide appeal. Very fittingly it is dedicated to Geoffrey Jefferson, who has done so much for cerebral surgery in this country, and introduced by Norman Dott, whose contributions to this branch of surgery are equally well known. Mr. Rowbotham informs us at once that it has been written primarily for those who are responsible for the treatment of acute head injuries and have not received a special training in neurosurgery or neurology. This is well, because many cases of head injury must, in the first instance at least, come under the care of the general surgeon, and as the methods of neurosurgery become more widely known and practised by general surgeons so will more cases be more effectively dealt with in the all-important early phases. The author quotes his own experiences frankly but refers to many well-known authors, among whom, however, we regret to note the absence of Victor Horsley's name, which appears only as "Horsley's wax", and is neither mentioned in reference to his trephine, depicted on p. 139, nor in connection with gunshot fractures of the skull, a subject in which he carried out some of the early experimental work relating to injuries produced by rifle bullets. We should also like to have seen de Martel's name mentioned in connection with the use of local anæsthesia.

The book consists of seven chapters, in the first four of which are discussed the mechanism of head injuries and the pathology, diagnosis, and treatment of closed injuries; the remaining three deal with open or compound wounds of the head, the results of injury to special parts of the brain and skull, and the sequelæ of head injuries. In his discussion on cerebral œdema the author mentions finding a swollen brain at operation under local analgesia in cases of acute head injury, but pays scant attention to treatment by dehydration, and no reference is made to the papers of ardent advocates of this treatment in this country. Electro-encephalography is briefly discussed, but Rendle Short's important case, one of the first in this country to illustrate the value of this investigation in intracranial hæmorrhage, is not mentioned. We cannot agree with the statement (p. 191) that chronic osteomyelitis is common after penetrating head injuries and that for this reason wide excision of bone should be carried out. While, however, we may find odd features to thus criticize, the work as a whole calls for nothing but praise, especially for those sections dealing with surgical methods. These are particularly well illustrated and clearly described, and should prove most helpful to surgeons whose knowledge of such special procedures may be either limited or rather vague. The book is written in a style which is easy to read and is printed on glazed paper in a large attractive type. The text is not without lighter touches, as when the author, in discussing the demeanour of the patient who has suffered from a head injury, writes (p. 251): "Speed of undressing in my experience is a social distinction. A working man always does this slowly and often loses his stud, whereas a professional man undresses rapidly." Mr. Rowbotham is at his best when describing surgical procedures, particularly those relating to compound injuries, and herein lies the great merit of this book. It will make readily known to general surgeons methods which many have been slow to learn and apply to the treatment of such cases. Many bad results have in the past been due to lack of this knowledge, for no matter how diligently and carefully the methods of abdominal surgery are applied to the skull and its contents, the result of treatment by such methods can be little short of disastrous. The publishers as well as the author are to be congratulated on a volume which is authoritative and practical in outlook, easy to read, convenient in size, and attractive in appearance.

Cancer of the Face and Mouth. Diagnosis, Treatment, Surgical Repair. By VILRAY P. BLAIR, M.D., SHERWOOD MOORE, M.D., and LOUIS T. BYARS, M.D., of St. Louis. $10 \times 6\frac{3}{4}$ in. Pp. 599, with 260 illustrations and 64 plates. 1941. London: Henry Kimpton. 50s. net.

IT is usual in this country to leave the treatment of malignant growths in the face region to the general surgeon, the plastic surgeon being called in later to make good the defect.

There are many points in favour of this arrangement: the general surgeon may carry out his destructive operation radically, unhampered by thoughts of disfigurement or deformity, and a period of observation for recurrence is given before the case is submitted to the plastic surgeon to undergo an often long series of reconstructive procedures. It does not always ensure, however, that the defect is left in the optimum condition for reparative work, and in many instances material unaffected by the malignant condition is unnecessarily sacrificed.

There can be little doubt, therefore, that there is a field for the plastic surgeon who has sufficient experience of cancer cases to treat the case throughout. *Cancer of the Face and Mouth* is written by a team thoroughly conversant with all aspects of the problem. In many of the cases described it is evident that a considerable amount of immediate reparative work was justifiable and that this lessened the misery of the patient, whilst preparing the way for reconstruction.

In the discussion of guiding principles "selection of a rational plan of treatment" is suitably stressed.

One sees far too many cases in which a dozen or more plastic operations have been performed, for which less than half that number would have sufficed had sufficient time and thought been given to careful and rational planning.

The essential honesty of the authors is indicated by the inclusion of cases in which beautiful repair results have been followed by recurrence of the original disease and by the naive statement, "the vermilion lip margin is lipstick".

The suggestion that gouge grooves should be made in exposed skull *at the time of destruction* to encourage early formation of granulations is a most useful one.

There is a helpful discussion of "surface controlled deep-growing cancer" which must appeal to all who have done much work of this kind.

The section on parotid tumours, with its warning against incomplete removal of the small innocent-looking mixed parotid tumour, is timely: stress is laid on the importance of removing the tumour with its enclosing gland tissue.

Low dosage of radium, repeated, is advised for angioma, e.g., of the cheek.

There is a particularly readable and instructive chapter by Sherwood Moore on "Radiation", in which stress is laid on the need for close collaboration between surgeon and radiotherapist if the best interests of the patient are to be served. This is brought out when, in discussing the value of single massive doses of X-radiation, mention is made of "accessible structures and those *artificially made so*". This refers to a technique by which wounds were packed open, given X rays, and sutured from a few hours to a day later.

A statistical review of 780 cases of Ellis Fischel is supplied by Dr. E. L. Keyes.

The plastic surgeon will feel sympathy for the difficulties experienced in treating a contracted socket. The patience and perseverance of the authors are indicated by the seven finicky procedures employed before a normal-looking palpebral fissure and a socket capable of accommodating a prosthesis giving suitable prominence were achieved.

The advisability of "delaying" flaps is stressed, a point of great importance in all plastic work on older patients.

The book is the outcome of twenty years' experience of some 1500 cases. There are 260 photographic illustrations and 64 plates in which the procedures are beautifully and clearly shown in line drawings. The lettering on some of these is confusing, blending too readily with the lines of the drawings. We suspect that the flap figured in *Plate* 11 *A* has its pedicle tubed on the wrong shoulder, and in *Plate* 20 *C* a small flap *Y* is referred to in the legend as "*b*".

This book is one of outstanding merit which all surgeons will find a fund of interest and information.

BOOK NOTICES

[*The Editorial Committee acknowledge with thanks the receipt of the following volumes. A selection will be made from these for review, precedence being given to new books and to those having the greatest interest for our readers.*]

Epilepsy and Cerebral Localization. A Study of the Mechanism, Treatment and Prevention of Epileptic Seizures. By WILDER PENFIELD, Litt.B., M.D., D.Sc., and THEODORE C. ERICKSON, M.A., M.Sc., M.D., Ph.D. Chapter XIV, Electro-encephalography, by HERBERT H. JASPER, Ph.D.; Chapter XX, Psychological Studies of Patients with Epileptic Seizures, by M. R. HARROWER-ERICKSON, Ph.D. 10 × 6½ in. Pp. 623 + x, with 163 illustrations. 1942. London: Baillière, Tindall & Cox. 44s. net.

The Foot and Ankle. Their Injuries, Diseases, Deformities, and Disabilities, with Special Application to Military Practice. By PHILIP LEWIN, M.D., F.A.C.S. Second edition. 9¼ × 5⅞ in. Pp. 665, with 304 illustrations. 1941. London: Henry Kimpton. 42s. net.

Aids to Surgery. By CECIL A. JOLL, M.S. (Lond.), M.D. (Bris.), B.Sc. (Lond.), F.R.C.S., Senior Surgeon and Lecturer on Surgery to the Royal Free Hospital, etc.; and REGINALD C. B. LEDLIE, M.B., B.S. (Lond.), F.R.C.S. (Eng.), Surgeon to the Royal Cancer Hospital, etc. Seventh edition. 6¼ × 4 in. Pp. 654 + xi, with 58 illustrations. 1942. London: Baillière, Tindall & Cox. 10s. net.

The Life and Teaching of Sir William Macewen. A Chapter in the History of Surgery. By A. K. BOWMAN, Regional Medical Officer to the Department of Health for Scotland. 9⅝ × 6 in. Pp. 425 + xii. 1942. London, Edinburgh, Glasgow: William Hodge & Co. Ltd. 21s. net.

Demonstrations of Physical Signs in Clinical Surgery. By HAMILTON BAILEY, F.R.C.S. (Eng.), Surgeon, Royal Northern Hospital, London; etc. Eighth edition. 8⅞ × 6 in. Pp. 336 + xii, with 455 illustrations, a number of which are in colour. 1942. Bristol: John Wright & Sons Ltd. 25s. net.

Surgery of Modern Warfare. Edited by HAMILTON BAILEY, F.R.C.S., Surgeon, Royal Northern Hospital, London; etc. Second edition (complete in two volumes). 9¾ × 6⅝ in. Volume I. Compiled by 43 Contributors. Pp. 480 + xvi, with 375 illustrations, many in colour. 1942. Edinburgh: E. & S. Livingstone. 40s. net.

Synopsis of the Preparation and After-care of Surgical Patients. By HUGH C. ILGENFRITZ, A.B., M.D., RAWLEY M. PENICK, jun., Ph.B., M.D., F.A.C.S., and URBAN MAES, M.D., D.Sc., F.A.C.S., Louisiana State University of Medicine. 7¾ × 4¾ in. Pp. 532, with 55 illustrations. 1942. London: Henry Kimpton. 25s. net.